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APPLICATION FOR A SITE ASSIGNMENT FROM THE CITY OF BOSTON BOARD OF HEALTH AND HOSPITALS

PROPOSED CITY OF BOSTON WASTE-TO-ENERGY FACILITY
SOUTH BAY, SUFFOLK COUNTY, MASSACHUSETTS

ADDENDUM: QUALITATIVE AND QUANTITATIVE HEALTH RISK ASSESSMENT

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QUALITATIVE AND QUANTITATIVE HEALTH RISK ASSESSMENT

Airborne Emissions from the City of Boston Waste-to-Energy Facility

Environmental Health and Toxicology Group
Meta Systems Inc

November 7, 1986

7-868-8660 Telex 951280 METASYSTEM CAM

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I. INTRODUCTION:

Purpose of the Qualitative and Quantitative Health Risk Assessment of Airborne Emissions From the City of Boston Waste-to-Energy Facility

The purpose of this risk assessment is to explain and quantify the magnitude of risk to human health that may result from emissions from the City of Boston Waste-to-Energy Facility. Given emission estimates for the various pollutants, meteorological conditions and other information, it is possible to project ambient concentrations of each pollutant and the amount or "dose" an individual will receive over the course of a lifetime at this estimated level of exposure. The toxicological characteristics of the pollutants, such as carcinogenic potency, can be obtained from the literature on environmental and occupational health and toxicology. With this and the dose to the individual, an estimated probability that an individual will experience health problems due to facility emissions may be derived.

Once the ambient concentration of pollutants attributable to emissions has been estimated, it can be compared with the existing background levels of pollutants. The facility will not operate in a vacuum: it is important to determine whether operation will bring the existing levels of pollutants up to an unacceptable level, or will instead make a negligible contribution to background.

An important objective of any risk assessment is to allow the reader to appropriately evaluate health risks due to facility emissions, in part by comparison with the risks inherent in our daily lives. Without this orientation, the magnitude of health risks attributable to operation of the Facility, however elegantly derived, are meaningless. It is important to stress that the result of any risk assessment is simply an estimate of the magnitude of risk: it is not an exact or immutable quantity. Rather, it represents an upper estimate of health risk, given the

reasonable and conservative assumptions made, that may be ascribable to the operation of the Facility.

II. APPROACH IN THE EVALUATION OF EXPOSURE RELATED HEALTH HAZARDS

Our approach to the evaluation of potential health hazards involves selection of relevant and appropriate health endpoints, e.g. cancer, kidney dysfunction, etc. Health effects of interest occur after both acute and chronic exposures. Acute health effects typically occur after a large exposure of short duration. Since acute effects arise from exposures many orders of magnitude higher than the highest concentrations projected for the facility, they will not be part of the risk assessment except where used in the development of scaling factors or thresholds. Only chronic health effects, i.e., those due to long term, low level exposures, are considered. Of these, cancer is the most important. However, chronic long term exposure to low levels of the non-carcinogenic pollutants, such as mercury, lead and pentachlorophenol, may impose other kinds of health risks. These are also identified and evaluated.

How an individual is exposed to a substance, or the route of exposure, is important in evaluating health risks. Inhalation, ingestion, and absorption through the skin (dermal absorption) are all possible routes of exposure to airborne pollutants which can eventually deposit in soil and dust. However, all three routes of exposure may not be important for all pollutants. For example, because the gaseous pollutants do not deposit in dust, only inhalation is considered as the route of exposure. In general, different routes of exposure are considered based on the characteristics of the individual substances.

- For gaseous pollutants and some others, e.g., HCl, inhalation is the only route of exposure considered.

- For substances which are adsorbed to particulates, deposit in soil and street dust, and are persistent in soil but are not absorbed through the skin, inhalation of the airborne particulates and exposure due to soil ingestion are considered. We do not distinguish between street dirt, house dust and soil.

- For substances which are very persistent once deposited in soil and are known to be absorbed (or bioavailable) through the skin on contact with contaminated soil, e.g., the polychlorinated dibenzo(p)dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and polychlorinated biphenyls (PCBs), inhalation, ingestion and dermal absorption are the evaluated routes of exposure.

We utilized published accepted or recommended standards and guidelines in evaluating health risks of the regulated pollutants. For these, the Massachusetts Department of Environmental Quality Engineering (DEQE) has developed draft Acceptable Ambient Levels (AALs), and the Environmental Protection Agency (EPA) has developed National Ambient Air Quality Standards (NAAQS). These limits were set based on careful consideration of the health impacts of each pollutant. Unless a substance emitted from the Facility is expected to substantially exceed the Federal standard or State guideline, no in depth risk analysis is performed.

For the unregulated pollutants, regulatory information was used, if it was available, in addition to other information based on experimental, occupational and other studies. These include Occupational Safety and Health Administration (OSHA) Permissible Exposure Levels (PELs), American Conference of Governmental

Industrial Hygienists (ACGIH) Threshold Limit Values (TLVs), US EPA 2,3,7,8-TCDD Toxicity Equivalence Factors (TEF), EPA/ Carcinogen Assessment Group (CAG) unit risk factors, International Agency for Research on Cancer (IARC) evaluations, etc.

In lieu of generally accepted recommendations, or where data is incomplete, we evaluate the potential human health risk a pollutant may impose with respect to the extent that the facility may add to exposure due to existing background levels, if known. This is augmented with clinical information, and results from epidemiology, chronic bioassays, mutagenicity tests, and other relevant biological analyses, making note of limitations.

III. GENERAL ASSUMPTIONS USED IN THE RISK ANALYSIS

Any risk assessment is made in the interest of the public health, and must include a worst case exposure scenario as well as a more realistic but still conservative exposure scenario. This involves the appropriate choice of: air concentrations; deposition rates of the pollutants; and sensitive receptors which will reliably reflect the high end and a more realistic average on the risk scale. For this analysis, we have defined risk as the additional probability of developing cancer (or other health effects where appropriate) over the course of a lifetime (70 years) due to operation of the proposed facility. With respect to "sensitive receptors," no individual could be expected to spend an entire lifetime in a daycare center, hospital or employed at a facility located in an area of maximum concentration.

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The maximum projected emissions data was used to calculate air concentrations and particulate deposition rates of the various pollutants for two exposure scenarios. **CASE I**, the upper

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bound or worst case risk scenario, was calculated for the maximally exposed individual, born and living an entire lifetime at the area of highest pollutant concentration and deposition. The upper bound worst case is exactly that, under the worst of all possible conditions, and therefore very unlikely. **CASE II**, a realistic worst case risk scenario, was calculated for the individual exposed over a lifetime to average levels of ambient concentrations over all receptor points. It was assumed, for **CASE II** that the population is distributed uniformly around the receptor array used in air modeling. For the data and methodology used to arrive at an average annual concentration and deposition rate for the total receptor array, see Appendix A.

Once estimates of maximum and average air concentrations and deposition rates were calculated, assumptions were made concerning:

- how much pollutant-containing air an individual inhales over the course of a lifetime,
- how the pollutants which deposit and are persistent in soil and dust, accumulate over the lifetime of the facility and the individual,
- how much pollutant-containing soil is ingested over the course of a lifetime and,
- how much pollutant-containing dirt adheres to an individual's skin over the course of a lifetime.

What follows are descriptions of how these variables were modeled and estimated.

A. INHALATION

It is important to consider that, over the course of a lifetime, the human respiration rate changes appreciably. It can be seen from the following table that children inhale much more air per kilogram body weight (kg bw) than very young infants or adults. Since the amount of a substance an individual is exposed to, or dose, is evaluated on per kg bw basis, it is more accurate to normalize respiration rate to a kg bw rate. Thus it is assumed that the lifetime, normalized respiration rate is close to 25 m^3 per day for a 70 kg adult.

Human Respiratory Rate

According to ICRP analysis*, reference human respiration rates are as follows:

Reference Group	Respiration Rate m^3/day	Normalized Respiration Rate m^3/kg/day
New born (age 0, weight 3.5kg)	0.78	0.22
Infant (age 1, weight 10kg)	3.8	0.38
Child (age 10, weight 33kg)	15	0.45
Adult Male (age 20, weight 70kg)	23	0.33
Adult Female (age 28, weight 58 kg)	21	0.36

The time weighted average of the normalized respiration rates (25 m^3/day) takes into account the decrease in oxygen demand that occurs over the years between age 55-70.

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* ICRP, International Commission on Radiological Protection, 1975 'Report of the Task Group on Reference Man (No. 23)' Pergamon Press, New York, pp 11, 338-341, 358-360.

B. ACCUMULATION OF POLLUTANTS IN SOIL

Emission from the facility and the rate of deposition of particulate-bound pollutants in soil was assumed to be constant over the thirty year lifetime of the facility, consequently individuals are exposed to progressively higher soil concentrations of pollutants. Pollutant deposition was assumed to be constant according to an average deposition velocity of 0.42 cm/sec*, and to be distributed uniformly in soil to the depth of 1 cm. Therefore, pollutant accumulation was calculated as: (deposition rate) x (pollutant emission rate). Pollutant accumulation is converted to units of mass pollutant per mass soil by dividing accumulation by an assumed soil density of 1.6 g/cm^3.** This corresponds to a linear increase in accumulation concentration since both deposition rate and pollutant concentration in air are assumed constant. After the 30 year operating lifetime, pollutant concentration in soil was assumed to remain constant at the concentration achieved after 30 years accumulation.

The following figure shows the general relationship between the concentration of pollutant (lead, for this example) in soil at time (t) resulting from pollutant deposition and time.

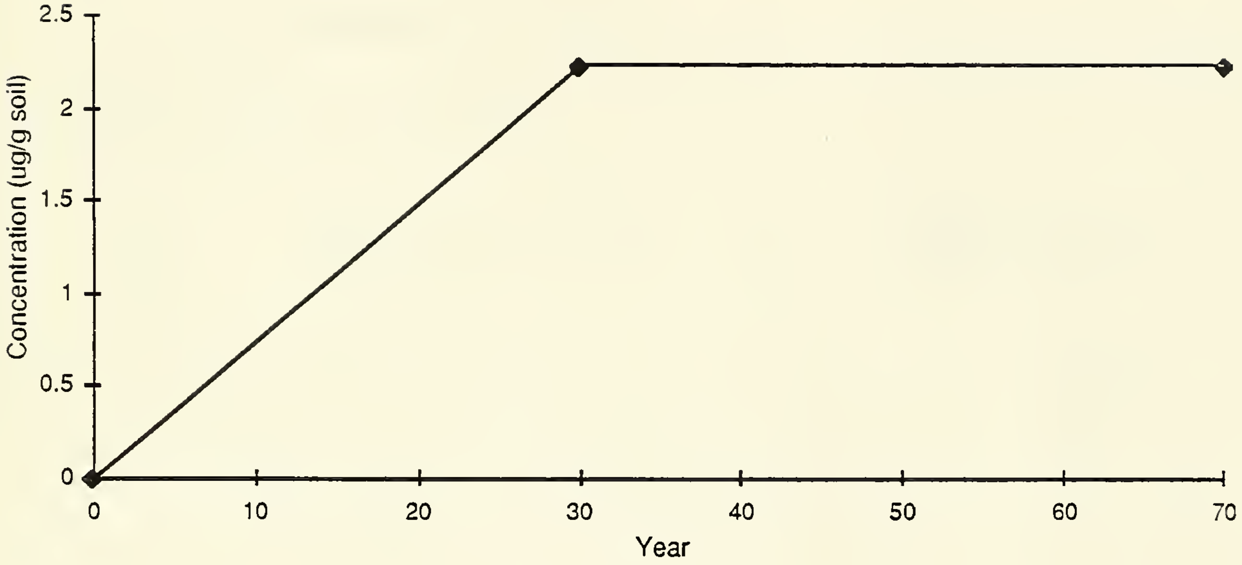
For risk assessment purposes, average daily exposure was estimated as the area under the curve from time t=0 to time t=70 divided by 70 years, where the area under the curve represents the time integral of concentration x time.

* CSI Resource Systems, Inc., September 29, 1986.

** Bouwer, H., Groundwater Hydrology, 1978

Soil Concentration of Lead due to Facility

Case I (Worst Case)



The assumptions concerning pollutant accumulation in soil were based on the proposition that no pollutant removal takes place. Removal mechanisms such as rainfall events or snow removal were not considered. Likewise, pollutant concentrations in air are average steady state concentrations: variations in pollutant concentration due to changes in weather patterns or other factors were considered only to calculate an average concentration.

1. SOIL EXPOSURE: INGESTION

Estimates of exposure to pollutants via ingestion of soil were calculated as follows:

$$\sum_{t=0}^{70 \times 365} \begin{array}{l}\text{Pollutant}\\ \text{Accumulation in Soil}\\ \text{at Time (t)}\end{array} \quad \times \quad \begin{array}{l}\text{Human Soil}\\ \text{Ingestion Rate}\\ \text{at Time (t)}\end{array}$$

Soil ingestion was assumed to vary with age as shown[*]:

Age	Ingestion Rate
0-1.5 years	1 mg soil/day
1.5-3.5 years	100 mg soil/day for 9 months/year
3.5+ years	1 mg soil/day

Pollutant intake (ug/day) via ingestion of soil is therefore calculated as:

$$\begin{array}{l}\text{Pollutant}\\ \text{Accumulation}\\ \text{(ug pollutant/g soil)}\end{array} \quad \times \quad \begin{array}{l}\text{Soil}\\ \text{Ingestion Rate}\\ \text{(g soil/day)}\end{array}$$

---

[*] Paustenbach, *et al*, 1986

2. SOIL EXPOSURE: DERMAL CONTACT

It can be assumed that high exposure occurs during contact with soil, however since absorption of pollutants is not instantaneous, pollutant exposure for risk analysis purposes was calculated based on the amount of soil adhering to skin after contact.

A study by Roels et al (1980) indicated about 0.5 mg of soil per cm^2 of skin adheres to a child's hand after playing in and around the home. Using tables of values for human skin surface area (Snyder, 1975), daily exposure due to dermal contact with soils can be estimated assuming uniform distribution of soil over the entire skin surface by multiplying pollutant concentration in soil (steady state soil contaminant concentration) by the amount of dirt on the skin and the total skin surface area.

Dermal Exposure Assumptions Used in Calculations*:

- 0.5 mg soil/cm^2 skin
- 1400 cm^2 exposed surface
- 8 hour/day, 90 days/year

* Paustenbach, et al., 1986

In examining the health effects of each pollutant, adjustments were made in the exposure estimates to calculate as realistic an actual dose as possible. Appropriate biological parameters must be incorporated in the estimate. For example, if X mg of pollutant A is ingested in soil over a lifetime, but it is known from human information and/or animal experiments that only 1% is actually absorbed during transit through the gastrointestinal tract (bioavailable), then the actual dose ingested over a lifetime is very much smaller: (.01)(X) mg.
Where bioavailability and other important information is unknown, it is in the interest of the public health to assume the worst

case: that 100% of the inhaled or ingested dose is bioavailable. It is important to keep in mind that these assumptions probably represent a significant overestimation of actual risk.

Where several pollutants impose risks for the same health effect, e.g., cancer or nephrotoxicity, the cumulative risk is presented.

IV. RISK ASSESSMENT

A. REGULATED POLLUTANTS

1. GENERAL CONSIDERATIONS

The maximum average annual ambient concentrations of the regulated pollutants present in emissions in non-negligible amounts are presented in the table on page 13. To provide a more complete environmental picture, the existing levels measured in Boston have been provided if available in the column marked Background (CSI 1986). It can be seen that the concentrations resulting from Facility emission of particulates, SO_2, NO_2, CO, Be, and H_2SO_4 are very low, between a factor of 10 to 1000 lower than either DEQE AALs or NAAQS'. All are below current Boston background pollutant levels. The health effects of these substances at the projected levels are negligible and will not be considered in this assessment. Of the regulated pollutants, lead and mercury present a different concern and will be discussed on an individual basis.

Lead emissions projected for the Facility are well within the recognized safe limits: even the maximum projected concentration for lead is ten times lower than the DEQE AAL, and the worst case annual concentration in air is only one percent of measured ambient air lead levels in and around Boston. However, since Boston has a high background level of lead in urban soil, any contribution from the facility to existing lead levels requires closer scrutiny. Therefore, since ingestion of lead in

soil is an important route of exposure, the cumulative contribution of airborne particulate lead to dirt and soil was examined, in addition to inhalation of airborne lead.

Kidney function is also adversely affected by lead and mercury. The relative contribution of these and other heavy metals to the overall toxicity to kidneys, or nephrotoxicity, of Facility emissions is discussed in following sections.

Average Maximum Annual Ambient Concentrations of Regulated Pollutants

		Concentration (ug/m^3)			
Pollutants	Averaging Period	Background Level	Maximum Concentration[a]	DEQE AAL	NAAQS Standard
Regulated Particulates	24-hour	132	1.4		260(150[b])
	Annual	57	1.8×10^{-1}		75
SO_2	3-hour	427	21.0		1300[b]
	24-hour	187	8.1		365
	Annual	47	1.0		80
NOx (as NO_2)	1-hour		64.2[c]		
	24-hour				
	Annual	90.4	1.8[c]		100
CO	1-hour	13,770	64.3[d]		40000
	8-hour	9,677	21.6[d]		10000
	Annual		4.6×10^{-1}[d]		
Lead (Pb)	24-hour	8.7×10^{-1}	7.0×10^{-2}	6.8×10^{-1}	prop
	3-month	5.6×10^{-1}	1.0×10^{-2}		1.5 → 0.5
	Annual	3.1×10^{-1}	9.0×10^{-3}		
Mercury (Hg)	24-hour		1.4 1.0×10^{-2}		
	Annual		1.9 2.0×10^{-3}	3.3×10^{-1}[e]	
Beryllium (Be)	24-hour		2.7×10^{-5}	7.0×10^{-4}	
	Annual		3.5×10^{-6}		
F- (as HF)	24-hour		8.0×10^{-2}	6.8	
	Annual		1.0×10^{-2}		
H_2SO_4 (mist)	24-hour		2.5×10^{-1}	13.6	
	Annual		3.0×10^{-2}		

[a] Maximum concentration is calculated as X/Q (s/m^3) multiplied by emission rate (g/s), where X/Q represents maximum annual concentration rates calculated at each receptor point for the entire receptor array, based on a normalized emission rate (1.0 g/s).

[b] Value represents secondary ambient air quality standard.

[c] Maximum NO_2 concentration assumes 81% conversion of NO_x to NO_2.

[d] Maximum CO concentration based on 400 ppm (1-hour average) and 100 ppm (24-hour average) flue gas concentrations.

[e] New York State Interim annual average AAL derived from ACGIH TWA-TLU. See NYS Department of Environmental Conservation, 1985.

2. SPECIAL CONSIDERATIONS: LEAD AND MERCURY

LEAD

The acute toxicity effects of lead occur at levels orders of magnitude higher than projected in emissions from the Facility, and were not considered.

The chronic toxic effects of low level lead exposure are chronic nephropathy, central and peripheral neuropathy, and diminution of fertility. Carcinogenesis studies are generally inconclusive. Therefore, chronic toxic effects were considered in the risk assessment, not carcinogenesis.

The following tables of information on lead were abstracted from IARC (IARC, 1980) and are provided to orient the reader with respect to regulatory information, and typical background levels of lead elsewhere in the US and the world.

Lead Drinking Water Standards

100 ug/l, WHO international standard, 1971
50 ug/l, US EPA, 1976
100 ug/l, Japanese Ministry of Health & Welfare, 1978
50 ug/l European Economic Community, 1975

Occupational and Ambient Standards and Recommendations

1.5 ug/m^3, US EPA, 1978, ambient 3-month air quality standard (current)
50 ug/m^3, average 8 hour exposure, OSHA, 1979
500 ug/l, maximum blood level, OSHA, 1979
200 ug/l, maximum blood level, general population, EEC, 1978
350 ug/l, maximum blood level, individual, EEC, 1978
150 ug/m^3 , NIOSH

Air Levels

0.0001-0.001 ug/m^3, areas remote from civilization
4-5 ug/m^3, Los Angeles, 1971
2.0 ug/m^3, Los Angeles, 1974
54.3 ug/m^3, L.A major highway, morning rush hour, 1972
1.1 ug/m^3, average urban
0.21 ug/m^3, average suburban
0.02 ug/m^3, remote

Cigarette Smoke

10.4-12.15 ug/cigarette, approx. 2% available in smoke
5 ug/pack could be absorbed by smoker

Water

1-10 ug/l, global lakes & rivers
1-60 ug/l groundwater
0.08-0.4 ug/l coastal seawater
13.1 ug/l average residential tap water from 969 water systems in the US

Food (fresh wt)

1500 ug/kg, condiments
200 - 2500 ug/kg, seafood
0-370 ug/kg meat & eggs
0-11390 ug/kg, grain
0-1300 ug/kg, vegetables
20-40 ug/l, milk
100-500 ug/day, adult dietary intake
130 ug/day, children <3 yrs. dietary intake

Bioavailability

83% GI absorption in young rats

16% GI absorption in mature adult rats

64.5% GI absorption in young monkeys

3.2% GI absorption in mature adult monkeys

41.5% GI absorption in human infants

10-11.5% GI absorption in human adult

Toxicity Data

2,000,000 ug/kg bw lead chloride oral LD50 in guinea-pigs

110,000 ug/kg bw tetramethyl lead oral LD50 in rats

400 ug/l, blood level associated with encephalopathy, muscular weakness, and peripheral neuropathy in human adults

700 ug/l, blood level associated with chronic, irreversible nephropathy in human adults

1200-1400 ug/l, blood levels associated with renal and hepatic damage in human adolescents

Of all population groups, young children are the most sensitive to the toxic effects of lead. In areas of the city where the background and existing body burden of lead is already high, the health impact of additional lead due to operation of the Facility is the issue of greatest concern. Therefore, two additional risk scenarios were developed using the average maximum and mean soil lead obtained by the Suffolk County Extension Service from Emergency Lead Poisoning Areas (ELPAs), and sample data from Boston and surrounding communities. The ELPA data was derived from samples of soil collected at the homes of lead poisoned children, generally within three feet of the houses to a depth of 1-2 inches. The rest of the samples were mostly from gardens to a depth of 6 inches. Lead levels decline with depth and distance from lead painted buildings. The two lists of soil lead test results, which have been kindly provided by Ronald

R. Jones, of the City of Boston Department of Health and Hospitals, are attached as Appendix B.

The following table shows the percent contribution lead emissions projected for the Facility will make to existing air and soil lead background.

Background Boston Air and Soil Lead Levels and Projected Facility Lead Contribution

	Case I: Worst Case			**Case II: Average**		
	Back-Ground	Additional Lead due to Facility	% Change	Back-Ground	Additional Lead due to Facility	% Change
Air	3.1×10^{-1} ug/m^3	9.0×10^{-3} ug/m^3	2.8	3.1×10^{-1} ug/m^3	1.0×10^{-3} ug/m^3	3.2×10^{-1}
Soil	5183 ug/g soil	1.8 ug/g soil*	3.4×10^{-2}	2006 ug/g	1.8×10^{-1} ug/g soil*	9.3×10^{-3}

* Soil concentration due to facility operation calculated as described above.

The most useful analysis in the case of lead, is to examine the relative contribution of the Facility lead emissions to blood lead levels in children in both maximally exposed individuals living in highest lead background areas, and individuals living in average high background exposed to average lead levels.

To calculate the contributions of air and soil lead to blood levels in children, air and soil concentrations were multiplied by empirically derived bioconcentration factors (EPA, 1984; Elwood, 1986). It can be seen from the data tabulated on page 18 that the contribution of lead in emissions is not significant in

relation to background. The facility will not contribute a significant amount to total blood lead levels in average or maximally exposed high risk children.

Relative Contribution of Facility Lead Emissions to Lead Levels in Boston High Risk Children

ir Concentration ug/m^3

	Back-ground	Addition due to Facility	Air/Blood Relation*	Concentration in Blood due to:		
				Background	Facility	% Increase
ase I	0.310	0.009	1.96	0.608	0.018	2.82
ase II	0.310	0.001	1.96	0.608	0.002	0.32

oil Concentration (ug/g)

	Back-ground	Addition due to Facility	Air/Blood Relation*	Concentration in Blood due to:		
				Background	Facility	% Increase
ase I	5183	1.8	$1.8\text{x}10^{-3}$	9.3	0.003	0.034
ase II	2006	0.18	$1.8\text{x}10^{-3}$	3.6	$3.3\text{x}10^{-4}$	0.009

* Elwood, Peter C., The Sources of Lead in Blood: A Critical Review; the Science of the Total Environment, 52 (1986) 1-23. The Air/Blood relationship is in units of ug Pb/dl blood per ug Pb/m^3 air.

A "draft final" criteria document is expected to be published in the Federal Register within the next several weeks suggesting that the maximum quarterly average standard for lead in the air be lowered from 1.5 ug/m^3 to 0.5 ug/m^3 (Washington Newsletter (1986) Chemical Week, November 5, p48). The lower standard of 0.5 ug/m^3 would still be 55 times higher than the lead concentration predicted for the worst case scenario. Even on top of existing background ambient levels will not exceed the 0.5 ug/m^3) standard.

It must be noted that lead represents a very real problem for children living in high risk areas in and around Boston. The proposed facility, however, will not add significantly to the overall lead burden.

The contribution of lead to cumulative heavy metal nephrotoxicity is discussed in detail on page 65.

MERCURY

The toxic effects of mercury range from those encountered at very low chronic exposures; erethism and paresthesia (irritability and abnormal prickling or itching sensations, respectively) to ataxia, dysarthria, deafness and death from much higher acute exposure. The central nervous system and kidney are target organs in people who have been chronically exposed.

To give the reader a regulatory perspective on mercury, the following guidelines are summarized from the Handbook of Toxic and Hazardous Chemicals and Carcinogens (Sittig, 1985).

Guidance in Air:
100 ug/m^3, Federal Ceiling
50 ug/m^3, NIOSH Recommendation
50 ug/m^3, ACGIH TWA

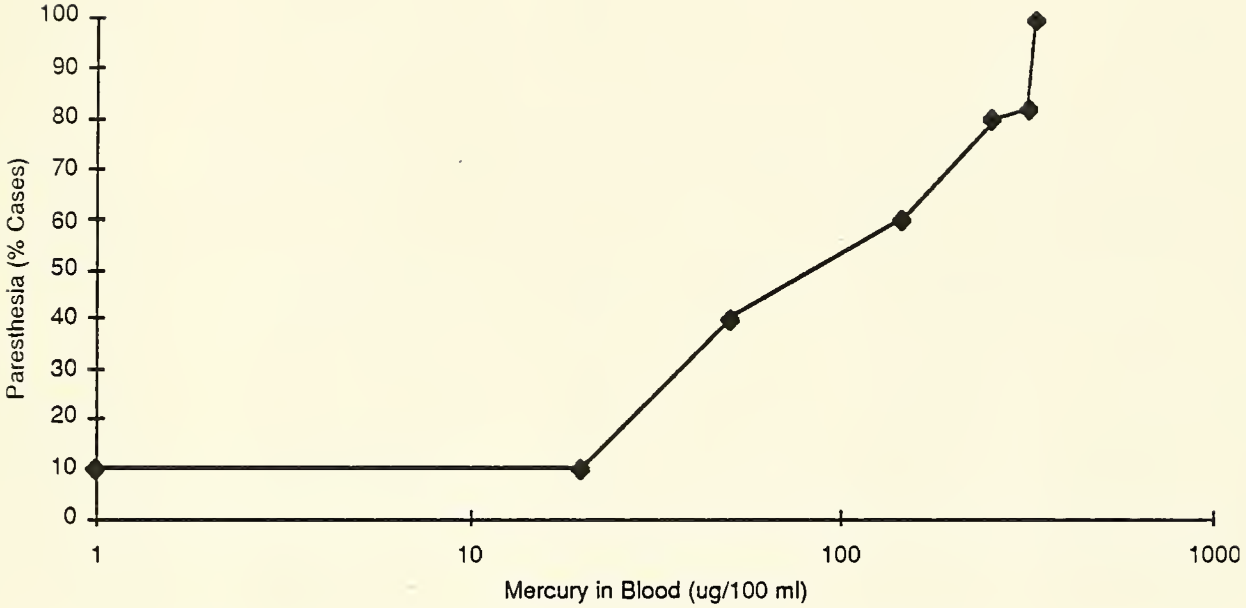
The exposure dose-response relationship between the amount of mercury inhaled and measurable amounts of mercury in the blood have been reported. Studies using occupationally exposed individuals have shown that as the air concentration of mercury rises above 100 ug/m^3, the frequency of erethism (one of the classic early signs of mercury intoxication) increases (Hammond, et al, 1980).

Paresthesia is also an early sign of mercury intoxication. The relationship between blood mercury levels and the appearance of paresthesia in exposed populations has been defined. Below appears a figure representing the relationship between appearance of paresthesia with blood mercury levels.

For risk assessment purposes, it was assumed that chronic long term exposure to air levels of mercury at 100 ug/m^3 will produce blood mercury levels of 250 ug/l and be just below the threshold for induction of detectable mercury toxicity. Mercury and certain other metals have been associated with renal dysfunction and will be analyzed with respect to this effect both on an individual and cumulative basis. Because paresthesia appears at blood levels of mercury lower than proteinuria, an indicator of nephrotoxicity (Hammond, 1980), it was conservatively assumed that this is also the threshold for nephrotoxicity.

It was assumed that all mercury in emissions is in the vapor phase, that the important route of exposure is inhalation, and that 100% of inhaled mercury is bioavailable. The level of mercury in blood and percent of the adverse effects threshold were calculated for the average and maximally exposed individual and are tabulated below.

Dose-response Relationship for Methyl Mercury in Blood



From Bakir, F., *et al.* Methyl-mercury poisoning in Iraq. An Interuniversity report. *Science*, **181**:230-41,1973.

Mercury Emissions and Projected Corresponding Blood Mercury Levels

Concentration

	Air ug/m^3	Blood ug/l	Blood Threshold ug/l	% of Threshold
Case I	0.002	0.005	250	0.002
Case II	0.0002	0.0005	250	0.0002

It can be seen from the data in the preceding table that the rise in blood levels of mercury of even the maximally exposed individual will account for only 2 X 10^{-3}% of the nephrotoxic blood mercury threshold. Looking at this a different way, the New York State Department of Environmental Conservation (NYS DEC) annual average AAL for mercury in air is 0.33 ug/m^3; it has been formulated with added safety factors to protect the most sensitive individual, (i.e. children, elderly, or infirmed), in the population breathing this level daily for a lifetime. The highest mercury concentrations are more than 160 times lower, and the average concentrations are more than 1600 times lower than the NYS DEC annual average AAL for mercury.

Based on the information stated above, significant adverse health effects from mercury in emissions are not anticipated.

B. NON-REGULATED POLLUTANTS

All non-regulated pollutants present in emissions were addressed and considered in the risk assessment. Substances which have been determined to be toxicologically important were subjected to a detailed risk analysis.

1. TRACE METALS

The trace metals were addressed on an individual basis and a carcinogenic risk analysis was performed for arsenic, cadmium, chromium, and nickel. Potential additivity of the nephrotoxicity of lead, mercury and cadmium was also quantified and discussed.

The table on page 23 shows the projected air concentrations of trace metals from emissions calculated for CASE I, the worst case exposure scenario and CASE II the average exposure scenario.

Estimated Air Concentrations for Trace Metals

	Concentration (ug/m^3)	
	Case I*	Case II**
Trace Metals		
Antimony	4.3×10^{-4}	4.5×10^{-5}
Arsenic	7.0×10^{-5}	7.2×10^{-6}
Barium	1.3×10^{-3}	1.3×10^{-4}
Cadmium	3.4×10^{-4}	3.5×10^{-5}
Chromium	1.7×10^{-4}	1.7×10^{-5}
Cobalt	3.4×10^{-5}	3.4×10^{-6}
Copper	3.6×10^{-4}	3.7×10^{-5}
Nickel	4.5×10^{-5}	4.7×10^{-6}
Selenium	2.1×10^{-4}	2.2×10^{-5}
Zinc	2.5×10^{-2}	2.6×10^{-3}

* Worst Case Annual Concentration calculated as X/Q (s/m^3) multiplied by emission rate (g/s), where X/Q is based on a normalized emission rate (1.0 g/s) for receptor location distance 0.9km, 110 degrees.

** Average Annual concentration calculated as X/Q (s/m^3) multiplied by emission rate (g/s), where X/Q is the average of X/Q for all receptors.

ANTIMONY

Because antimony and arsenic appear together so often, it is difficult to distinguish their respective health effects. Antimony and its salts have been used therapeutically as emetics, expectorants and parasiticides. Antimony has been ingested in cough syrups at doses of 1,000 - 8,000 ug two or three times daily, and a total injected dose for the treatment of schistosomiasis can range up to 2,500,000 ug over a 30 day course. It is still used in the treatment of leishmaniasis at a dose of 20 mg/kg bw per day for 30 days. Patients treated thus have shown no appreciable differences in pre- and post-treatment renal function test results (Jolliffe, 1985).

Antimony is not considered a human carcinogen by IARC, and at high enough concentrations it acts primarily as an irritant. Concentrations of antimony necessary to cause acute adverse health effects are many hundred times higher than that projected for Facility emissions.

Antimony is a common urban pollutant, and occurrence and regulatory information from various sources are provided below.

Antimony in Air:
Average Urban Atmospheric levels 1 X 10^{-3} ug/m^3.
NYS DEC AAL 0.67 ug/m^3
OSHA TLV 500 ug/m^3

Antimony in Water:
EPA ambient limit 7 ug/l

The highest concentration of antimony in air (table on page 23), is seen to be 0.06% of the AAL for New York State; the average concentration is even smaller: 0.006%.

Significant adverse health effects attributable to antimony toxicity from projected Facility emissions are not anticipated.

ARSENIC

The acute toxic effects of Arsenic are observed at exposure levels so much higher than those expected from the Facility, only health effects associated with low level chronic exposure was considered in the risk assessment.

The following tables provide relevant information on toxicity and other health effect endpoints associated with exposure to Arsenic for comparison with the calculated maximum and average air concentrations projected for Facility emissions; included are various guidance levels, toxicity, occupational standards, ambient air and water levels; and concentrations of arsenic in media such as food and cigarettes.

Acute Toxicity Data

Chemical	LD_{50} (oral)	LD_{50} (ip)
As[V] (Arsenate)	100mg/kg (rats and mice)	14-18mg/kg (rats)
As[III] (Arsenite)	10mg/kg (rats and mice)	5mg/kg (mice)

Occupational Exposure Limits and Recommended Maximum Concentrations for Arsenic

Agency	Level	Additional Notes
NIOSH (1973)	50ug/m^3	proposed, 8-hour TWA
NIOSH (1975)	2ug/m^3	proposed
OSHA (1975)	4ug/m^3	inorganic, proposed, 8-hour TWA
OSHA (1978)	10ug/m^3	inorganic 8-Hour TWA
	0.67ug/m^3	NYS DEC AAL (current)
ACGIH (1983/4)	200ug/m^3	As + soluble compounds

Levels of Arsenic in Ambient Air*

Concentration	Comment
0.03 ug/m^3	Average at 133 U.S. Stations 1950-1964

Levels of Arsenic in Water*

Concentration	Comment
10-50 ug/kg	727 Samples of U.S. surface water
0.82-17ug/l	Rain; US
198,000-243,000ug/l	Lake; Searles Lake, CA
0-200ug/l	California
	U.S. average
0.2-117ug/l	
100-6000ug/l	River
<10-1100ug/l	Creek
0.25-180ug/l	USA average

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* From: IARC (1980) IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Some Metals and Metallic Compounds Vol 23, IARC, Lyon.

Drinking Water Standards

EPA Federal Standard (current)	50ug/l	under review; A level of 0.002 ug/l corresponds to a risk of 1×10^{-5}
S. Africa	50ug/l	
WHO (1980)	50ug/l	
W. Germany	50mg/l	

Arsenic Intakes from Food and Cigarettes*

Food:	Concentration or Amount	Comment
	1,400 ug/kg	Fish
	50-160 ug/kg	wet weight in average diet
	137-330 ug	average adult intake from food

Cigarettes:	Concentration or Amount
	7.7 ug/gram tobacco

* From: IARC (1980) IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Some Metals and Metallic Compounds Vol 23, IARC, Lyon.

In considering the most important routes of exposure to arsenic, data from studies of exposures to residents (mostly children) living in the vicinity of smelters, and occupational exposures of workers have been reviewed. In these settings, the levels of arsenic in the air and soil make ingestion and dermal contact important for the assessment of irritant effects. However, these levels greatly exceed the projected Facility arsenic emissions, risk assessment will be performed for effects due to chronic exposure. Lung cancer is the most important health effect known to be related to chronic arsenic exposure. For the purpose of estimating the excess lifetime risk of cancer due to the arsenic in Facility emissions, we used the EPA CAG

95% confidence upper bound limit for the unit risk of lung cancer; 15 $(mg/kg/day)^{-1}$. The unit risk factor, or potency slope is an estimate of carcinogenic potency based on animal and human data. It is multiplied by the daily intake per kg bw to yield the estimated excess lifetime cancer risk. Cancer risk analysis for arsenic addressed only inhalation as the route of exposure.

Arsenic commonly exists in two valence states; As(III) and As(IV). It is generally accepted that the trivalent arsenic compounds are the more important toxicologically (IARC, 1980). As there is no way to reliably measure the ratio of As(III) to As(IV) in emissions, it was assumed, conservatively, that all arsenic in Facility emissions exists as As(III).

Data from animal studies indicate that arsenic is readily absorbed from the respiratory tract (92% in rats). Therefore, it is not unreasonable to assume that 100% of the arsenic inhaled is bioavailable. Tabulated below are the total daily intake of arsenic and calculated excess lifetime risk of cancer.

Lifetime Daily Intake of Arsenic and

Estimated Excess Lifetime Cancer Risk

	Daily Intake (mg/kg/day)	EPA/CAG Potency Slope $(mg/kg/day)^{-1}$	Excess Lifetime Cancer Risk
CASE I	2.5×10^{-8}	15	3.8×10^{-7}
CASE II	2.6×10^{-9}	15	3.9×10^{-8}

The estimated upper bound on risk of cancer over a lifetime at the projected arsenic concentrations is less than 4 in ten million and less than 4 in one hundred million for the worst and average exposure scenarios respectively.

BARIUM

Barium compounds are generally water soluble. The most notable exception is $BaSO_4$: the essential ingredient in the "barium swallow" or enema concoction used for X-ray visualization of corresponding portions of the gastrointestinal tract. Barium is not considered a carcinogen. The Federal standard and ACGIH value for permissible levels of soluble barium compounds in air is 500 ug/m^3. For safe levels in drinking water the National Academy of Science/ National Research Council (NAS/NRC) has recommended 2000 ug/l for adults and 1000 ug/l for children (Sittig, 1984). The highest concentration of barium in air is not expected to exceed 1.3×10^{-3} ug/m^3 and more realistically 1.3×10^{-4}. This is almost 400,000 and 4 million times less than the Federal Standard respectively. Therefore a detailed risk assessment for barium was not performed.

CADMIUM

Cadmium has been known to cause a variety of acute and chronic effects involving the kidneys, blood, respiratory system, and prostate. Because the levels of cadmium associated with acute effects vary between hundreds and thousands of times higher than the levels anticipated in emissions from Facility, these were be considered in the health risk assessment. The health effects of primary concern are those associated with low level chronic exposure; renal tubular dysfunction and lung cancer.

The following tables are provided for comparison with the projected cadmium concentrations from Facility emissions. They contain relevant information on toxicity, carcinogenicity and other health effect endpoints; various guidance levels; and ambient concentrations of cadmium in media such as air, food, water, cigarettes, etc.

Occupational Exposure Limits and Recommended Maximum Concentrations for Cadmium

Agency	Airborne Level	Additional Notes
OSHA Federal Standard (current)	100 ug/m^3	8 hour TWA
ACGIH (1983/84)	50 ug/m^3	8 hour TWA. 200 ug/m^3 Short Term Exposure Limit (STEL)
NIOSH	40 ug/m^3	8 hour TWA
WHO (1980)	10 ug/m^3	8 hour TWA. 200 ug/m^3 ceiling for 5 min.
Massachusetts DEQE Acceptable Ambient Level (AAL)	0.005	the AAL is not exceeded in Boston

Cadmium Concentrations in Air in the US[*]

Area	Concentrations (ug/m^3)	Comment
Remote:		
Nebraska	0.0057	
Rural:		
New York State	0.003	Tuxedo, NY
U.S.	0.004-0.012	30 sites
U.S.	0.0037	farm site
Urban:		
New York City	0.014-0.023	
U.S.	0.004-0.015	6 largest cities, annual averages
Industrial Areas:		
U.S.	0.12	El Paso Donora
U.S.	0.023-0.105	6 worst urban areas, annual averages

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[*] Data abstracted from: "Cadmium in the Environment and its Significance to Man," Pollution Paper No. 17 (1980). H.M. Stationary Office, London.

Cadmium Concentrations in Drinking Water and in Cigarettes

Drinking Water *	Comment
1.3 ug/l	average of data from 969 U.S. cities
Drinking Water Standards/ Recommended Maxima	
50 ug/l	S. African Bureau of Standards
10 ug/l	WHO
6 ug/l	West Germany
Cigarettes **	
1.5-2.0 ug Cd/cigarette	

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* Abstracted from: McCabe, *et al*, (1970) *J. American Water Works Assoc*. *62*:670-687.

** Abstracted from: Meden *et al*, (1972) Distribution of Cadmium and Nickel of Tobacco During Cigarette Smoking. *Environ*. *Sci*. *Technol*. *6*:830.

Cadmium Intakes from Daily Diets

Country	ug/day
United States	38*
Canada	52
West Germany	48
Rumania	38-64
Czechoslovakia	60
Japan (unpolluted areas)	59
Sweden	16-19
Australia	30-50
New Zealand	21-27
United Kingdom	15-35

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* This estimate is the mean of 7 years of data from the FDA's Total Diet Study. Individual year's estimates ranged from 26 to 51 ug/day.

Since the nephrotoxic effects of cadmium exposure are associated with levels several hundred times higher than projected for the Facility, renal dysfunction effects of cadmium and all the heavy metals are discussed on a cumulative basis on page 65.

It has been demonstrated, using studies of human occupational exposures in smelters, that the important route of exposure is inhalation. The bioavailability of cadmium has been reported to be 25% for inhalation (Friberg, et al, 1974, Cadmium in the Environment, CRC Press, Cleveland Ohio). For the risk assessment, it was assumed, conservatively that 50% of inhaled cadmium is bioavailable.

The lifetime carcinogenic risk of cadmium exposure is estimated and tabulated below using the projected maximum and average annual concentration in Facility emissions, and the EPA/CAG estimate for the upper 95% confidence limit for the unit risk of carcinogenesis due to cadmium: 6.1 $(mg/kg/day).^{-1}$

Lifetime Daily Intake of Cadmium and Estimated Excess Lifetime Cancer Risk

	Daily Intake (mg/kg/day)	EPA/CAG Potency Slope $(mg/kg/day)^{-1}$	Excess Lifetime Cancer Risk
CASE I	6.0×10^{-8}	6.1	3.7×10^{-7}
CASE II	6.3×10^{-9}	6.1	3.8×10^{-8}

It can be seen from the table that the excess lifetime risk of cancer due to cadmium emissions is less than 4 in ten million at worst, and probably closer to 4 in one hundred million.

CHROMIUM

Although chromium is essential in human nutrition as a micro-nutrient, at high levels it is an irritant and a carcinogen. Minimum concentrations of chromium in air required to cause reversible upper respiratory tract irritation in humans are higher than 10 ug/m^3. Since this figure is almost 60,000 and 600,000 times higher than the worst case and average chromium levels projected respectively for Facility emissions (see the table on page 23), no toxic effects are expected from chromium exposure, or chronic effects other than cancer.

The following tables are provided for the purpose of comparison and contain information on occupational standards, levels of chromium in ambient air and chromium intakes in food, water and cigarettes.

Occupational Exposure Limits and Recommended Maximum Concentrations for Chromium

Agency	Airborne Level	Additional Notes
OSHA	1000ug/m^3	Chromium Metal
Federal Standard (current)	500ug/m^3	Chromium III compounds
	500ug/m^3	Chromium VI, water soluble
	1000ug/m^3	Chromium VI, water insoluble
NIOSH (1976)	1ug/m^3	Chromium VI, based on cancer
	25ug/m^3	Chromium VI, based on other health effects
	50ug/m^3	Chromium VI-ceiling
ACGIH (1983/4)	500ug/m^3	Chromium Metal
	500ug/m^3	Chromium II compounds
	50ug/m^3	Chromium III compounds
	50ug/m^3	Chromium, VI-water soluble
	50ug/m^3	Chromium, VI-water insoluble
	150ug/m^3	Chromyl Chloride
DEQE (current)	0.34ug/m^3	Total Chromium
	0.001ug/m^3	Chromium VI

Levels of Chromium in Ambient Air*

Concentration	Comment
0.01-0.003 ug/m^3	200 urban stations, 1960-1969 annual mean concentrations
less than 0.01 ug/m^3	non-urban areas

Daily Chromium Intake from Food and Water*

Food:	Concentration or Amount	Comment
	20-50 ug/kg	vegetables
	20 ug/kg	fruits
	40 ug/kg	grains and cereals
	10-40 ug	average adult daily intake from food
Water:	Concentration or Amount	Comment
	0.7-84 ug/l	15 North American rivers
	Not detectable - 36 ug/l	public water supply range
	0.43 ug/l	median level in public water supply
	4 ug	average adult daily intake from water
	170 ug/l trivalent	Federal Standard, E.P.A. (current)
	50 ug/l hexavalent	

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* From IARC (1980) IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans; *Some Metals and Metallic Compounds* Vol 23, IARC, Lyon.

Chromium exists most commonly in three valence states: Cr(II), Cr(III), and Cr(VI). It is generally accepted that the hexavalent form of Cr is more important toxicologically, it has been implicated in the induction of lung cancer in chromium workers (IARC, 1980).

Recent evidence suggests that Cr(VI) is rapidly reduced to Cr(III) in the presence of organic material. Given its non-volatility, any chromium emitted from the facility would be particulate bound, in close association with electron donating species. Thus, it is highly unlikely that any Cr(VI) would survive incinerator conditions (Ted Rains, National Bureau of Standards, Gaithersburg, MD, personal communication, 8/4/86). It should be noted that urban atmospheric chromium concentration are about 200 times higher than the expected worst case emissions. However, it was assumed for risk assessment purposes that 5% of total chromium emitted from Facility is Cr(VI).*

Studies involving workers exposed to chromium in plating, polishing and welding operations, have demonstrated that water-soluble chromates are readily absorbed from the respiratory tract, (IARC, 1980). In the risk assessment, it was assumed that the most important route of exposure to chromium is via inhalation, and 100% is bioavailable. This is most certainly overly conservative, as most Cr(VI) compounds are only slightly soluble and only a fraction of the total intake is retained.

For the purpose of calculating the excess lifetime risk of cancer due to exposure to chromium emitted from the Facility, the EPA/CAG unit risk calculated for hexavalent chromium, 41 $(mg/kg/day)^{-1}$, is used in the table below.

* CSI Resource Systems Inc., Sept. 29, 1986

Lifetime Daily Intake of Chromium and Estimated Excess Lifetime Cancer Risk

	Daily Intake (mg/kg/day)	EPA/CAG Carcinogenic Potency Slope $(mg/kg/day)^{-1}$	Excess Lifetime Cancer Risk
CASE I	3.0×10^{-9}	41	1.2×10^{-7}
CASE II	$3.0 \times {}^{-10}$	41	1.2×10^{-8}

It can be seen from the table that the worst and average case lifetime cancer risks are approximately one in ten million and one in a hundred million, respectively.

COBALT

Cobalt is an essential human micronutrient, it is a constituent of the vitamin B12 complex. The requirement for cobalt in the human diet as B12 is about 0.13 ug/day. The Interim Primary Drinking Water Regulations (USEPA) do not limit cobalt in drinking water, nor has the WHO recommended a limit on its International or European Standards. This may be because the maximum No Observable Adverse Effects Level (NOAEL) concentration is more than ten times higher than levels found in water supplies (Sittig, 1985). In air, the ACGIH proposed TWA is 50 ug/m^3. It can be seen from the table on page 23 that worst case air concentration of cobalt due to projected facility emissions is about one and a half million times lower than the proposed ACGIH standard, and almost 4,000 times lower than the minimum daily requirement for cobalt. As such, significant health problems due to cobalt emissions are not anticipated.

COPPER

Copper salts act primarily as irritants. The Federal Standard for copper in air is 1000 ug/m^3 and to protect human health, 1000 ug/l in water (Sittig, 1985). From the concentrations shown in the table on page 23 it can be seen that the level of copper projected for Facility emissions is almost three million times lower than the federal standard. In fact, the current Boston background level of copper in air is 0.060 ug/m^3, approximately 167 times higher than worst case air concentration estimated for facility emissions (CSI, 1986). As such, significant copper-related health risks are not anticipated.

NICKEL

The dietary intake of nickel, at about 300 ug per day, represents the most significant source of nickel for non-occupationally exposed individuals (Sittig, 1985). Nickel is commonly present in US air at levels of 0.034-0.46 ug/m^3 (Hammond et al, 1980). In Boston, the background concentration of nickel in air averages 0.049 ug/m^3 (CSI, 1986). The DEQE AAL for nickel is 0.002 ug/m^3. In as much as:

- the projected worst case concentration of nickel in air is, at 3.5 x 10^{-4} ug/m^3, (almost 18 times lower than the levels DEQE considers safe), and

- nickel is not a known nephrotoxin;

Risk assessment did not include the chronic toxic effects of nickel in Facility emissions. However, since inhaled nickel compounds have been implicated in the induction of lung cancer in occupationally exposed individuals, a carcinogenic risk assessment was performed using the EPA/CAG carcinogenic potency slope of 2.1 (mg/kg/day)$^{-1}$. In lieu of empirically obtained

bioavailability data, it was assumed that 100% of inhaled nickel is available. See the table below for the estimated lifetime excess risk of cancer attributable to nickel in Facility emissions.

Lifetime Daily Intake of Nickel and

Estimated Excess Lifetime Cancer Risk

	Daily Intake (mg/kg/day)	EPA/CAG Carcinogenic Potency Slope $(mg/kg/day)^{-1}$	Excess Lifetime Cancer Risk
CASE I	1.6×10^{-8}	2.1	3.4×10^{-8}
CASE II	1.7×10^{-9}	2.1	3.5×10^{-9}

It can be seen that the estimated lifetime excess risk of cancer attributable to nickel in Facility emissions is at worst, less than 4 in one hundred million, and closer to less than 4 in one billion.

SELENIUM

The role of selenium as an essential element in animal nutrition is well established; it has a significant role in the biosynthesis of coenzyme Q, and is an essential constituent of glutathione peroxidase (Hammond, et al, 1980). At chronic low or very high exposure levels, it is not known to be nephrotoxic, or carcinogenic. It is present in such over the counter anti-dandruff preparations as "Selsun Blue".

The DEQE AAL for selenium in air is 2.7 ug/m^3. Since the level considered acceptable by the DEQE is almost 1700 times higher than the worst case annual concentration estimated for Facility emissions, a risk assessment was not performed for selenium.

ZINC

Zinc is ubiquitous in biological systems and is considered essential in human nutrition. Human dwarfism and lack of sexual development have been related to zinc deficiency. Therapeutically, zinc compounds are used as topical astringents and dermal products, especially athletes foot and antidandruff preparations. It is estimated that the average daily intake of zinc primarily in the diet, is about 12,600 ug (Hammond, et al, 1980).

At low levels zinc is not known to be nephrotoxic or carcinogenic. The levels of zinc required to precipitate chronic toxic effects are several orders of magnitude higher than maximum concentrations projected from Facility emissions. In fact, the maximum worst case annual zinc concentration in air is 3.4 times smaller than existing background air levels of zinc in and around Boston (CSI, 1986). Significant health problems related to the zinc in Facility emissions are not anticipated.

2. ORGANICS AND ACIDS

This group of pollutants includes HCl, aldehydes organic acids, and the polycyclic aromatic hydrocarbons (PAH). The table on page 43 gives the concentrations in air of these pollutants calculated as described from projected Facility emission data.

Estimated Air Concentrations of the Organics and Acids:

HCl, Aldehydes, Organic Acids and PAH

	Concentration (ug/m^3)	
Pollutants	Case I*	Case II**
HCl	0.406	0.042
Aldehydes (as CH2O)	0.033	0.003
Organic Acids (as C2H4O2)	0.070	0.007
PAH:		
Pyrene	3.4×10^{-7}	3.5×10^{-8}
Fluoranthene	1.5×10^{-7}	1.6×10^{-8}
Phenanthrene	8.4×10^{-7}	8.6×10^{-8}
Benzo(a)pyrene***	2.1×10^{-8}	2.2×10^{-9}
Benzo(e)pyrene	2.1×10^{-8}	2.2×10^{-9}
Perylene	4.3×10^{-8}	4.5×10^{-9}
Coronene	4.3×10^{-8}	4.5×10^{-9}
Indino(123cd)-perylene	4.3×10^{-8}	4.5×10^{-9}
Benzo(b)-fluoranthene***	7.7×10^{-8}	7.9×10^{-9}
Benzo(a)-anthracene***	1.7×10^{-7}	1.8×10^{-8}
Benzo(ghi)-perylene	9.1×10^{-8}	9.3×10^{-9}
Total PAH	1.8×10^{-6}	1.9×10^{-7}

* Worst Case Annual Concentration calculated as X/Q (s/m^3) multiplied by emission rate (g/s), where X/Q is based on a normalized emission rate (1.0 g/s) for receptor location distance 0.9km, 110 degrees

** Average Annual Concentration calculated as X/Q (s/m^3) multiplied by emission rate (g/s), where X/Q is the average of X/Q for all receptors.

*** Carcinogenic PAH, IARC, 1982

HCl

The concentrations of HCl expected in air calculated from emissions projected for the Facility are shown in the previous table. The DEQE AAL for HCl in air based on a 24 hour measurement is 9.5 ug/m^3. The projected 24 hour maximum average concentration of HCl is 3.1 ug/m^3 for the worst case scenario and 0.3 ug/m^3 for an average concentration. The projected 24-hour air level is 3 times lower than the level which DEQE considers acceptable for 24 hour average concentrations. Significant toxic effects from HCl exposure attributable to projected Facility emissions alone are not anticipated.

ALDEHYDES

The levels of aldehydes in air projected for the Facility are calculated above in the table on page 43. The entire amount of emitted aldehydes were assumed, for risk assessment purposes, to be formaldehyde. This is actually a very conservative overestimate; the true formaldehyde content is probably closer to 50%. The ACGIH TWA for formaldehyde in air is 1500 ug/m^3 (Sittig, 1985). Projected concentrations are far below those necessary to induce acute or chronic toxicity effects. However, formaldehyde is a suspected human carcinogen and a risk assessment was performed using the EPA/CAG carcinogenic potency slope, 6.1 x 10^{-3} (mg/kg/day)$^{-1}$. The table on page 45 presents the lifetime daily intake of formaldehyde and the excess risk of cancer in an exposed individual's lifetime.

Lifetime Daily Intake of Formaldehyde and Estimated Excess Lifetime Cancer Risk

	Daily Intake (mg/kg/day)	EPA/CAG Carcinogenic Potency Slope $(mg/kg/day)^{-1}$	Excess Lifetime Cancer Risk
CASE I	1.2×10^{-5}	6.1×10^{-3}	7.2×10^{-8}
CASE II	1.1×10^{-6}	6.1×10^{-3}	6.5×10^{-9}

It can be seen from the worst case calculation that the cancer risk attributable to a lifetime spent exposed to the projected formaldehyde concentrations is smaller than one in ten million, and more likely less than 7 in one billion.

ORGANIC ACIDS

All the organic acids present in Facility emissions are assumed to be present as acetic acid. The concentrations of acetic acids in air projected for Facility are shown in the table on page 43. Acetic acid is a relatively benign substance, it is the essential constituent of vinegar. At appropriately high exposures, it acts as an irritant. The Federal standard for the permissible exposure limit in air is 25,000 ug/m^3. Concentrations of acetic acid in air due to Facility emissions are 357,000 and 3,570,000 times lower than the Federal standard, for the worst case and average air concentration scenarios respectively. Significant chronic toxicity effects attributable to Facility-generated acetic acid are not anticipated.

POLYCYCLIC AROMATIC HYDROCARBONS (PAH)

The various types of PAH expected to be present in Facility emissions, and their relative concentrations in air are shown in the table on page 43. The most important health effect endpoint for the PAH as a group is cancer. However, not all of the individual PAHs are known to be carcinogenic. Of the group, the

substances for which there is considered to be sufficient evidence of carcinogenicity in experimental animals are benzo[a]pyrene, benzo[b]fluoranthene and benzo[a]anthracene (IARC, 1982). Therefore, a carcinogenic risk assessment was performed for these three PAHs. Since benzo[a]pyrene is the most potent carcinogen of these, it was conservatively assumed that the others possess equivalent carcinogenic potency. Thus, the carcinogenic PAHs were assessed as a group using the EPA/CAG carcinogenic potency slope for benzo[a]pyrene. Since PAH are not persistent in the environment, inhalation was the evaluated route of exposure. It was assumed that 100% of all inhaled PAH are bioavailable. The table below gives the total daily intake of carcinogenic PAH and calculated excess lifetime risk of cancer.

Lifetime Daily Intake of Carcinogenic PAH and

Estimated Excess Lifetime Cancer Risk

	Daily Intake (mg/kg/day)	EPA/CAG Carcinogenic Potency Slope $(mg/kg/day)^{-1}$	Excess Lifetime Cancer Risk
CASE I	9.6×10^{-11}	11.5	1.1×10^{-9}
CASE II	1.0×10^{-11}	11.5	1.1×10^{-10}

It can be seen from the table above that the lifetime excess cancer risk is approximately one in a billion and one in ten billion for the worst case and average case respectively.

3. CHLORINATED ORGANICS

This group of pollutants includes the polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs), chlorobenzenes, chlorophenols, and the polychlorinated biphenyls (PCBs). The table on the following page gives the concentrations

in air of these pollutants calculated as described from projected Facility emission data.

Air Concentrations of Chlorinated Organics Due to Facility Emissions

	Concentration (ug/m^3)	
	Case I	Case II
2378-TCDD	1.6×10^{-9}	1.6×10^{-10}
Other TCDDs	2.2×10^{-8}	2.3×10^{-9}
2378-PeCDD	1.1×10^{-8}	1.1×10^{-9}
Other PeCDDs	3.2×10^{-8}	3.3×10^{-9}
2378-HxCDD	7.7×10^{-9}	8.0×10^{-10}
Other HxCDDs	5.5×10^{-8}	5.7×10^{-9}
2378-HpCDD	1.5×10^{-8}	1.5×10^{-9}
Other HpCDDs	1.5×10^{-8}	1.5×10^{-9}
OCDD	9.8×10^{-9}	1.0×10^{-9}
2378-TCDF	2.6×10^{-8}	2.6×10^{-9}
Other TCDFs	3.3×10^{-7}	3.4×10^{-8}
2378-PeCDF	3.8×10^{-8}	3.9×10^{-9}
Other PeCDFs	6.7×10^{-8}	6.9×10^{-9}
2378-HxCDF	2.6×10^{-8}	2.7×10^{-9}
Other HxCDFs	2.2×10^{-7}	2.2×10^{-8}
2378-HpCDF	1.5×10^{-8}	1.5×10^{-9}
Other HpCDFs	1.5×10^{-8}	1.5×10^{-9}
OCDF	2.4×10^{-9}	2.4×10^{-10}
PCB	1.7×10^{-7}	1.7×10^{-8}
124-Trichlorobenzene	1.3×10^{-6}	1.3×10^{-7}
Hexachlorobenzene	5.5×10^{-7}	5.7×10^{-8}
Total Chlorobenzenes	6.1×10^{-6}	6.2×10^{-7}
Tri Chlorophenol	4.8×10^{-6}	5.0×10^{-7}
Penta Chlorophenol	1.0×10^{-6}	1.1×10^{-7}
Total Chlorophenols	1.5×10^{-5}	1.5×10^{-6}

POLYCHLORINATED DIBENZO-p-DIOXINS AND DIBENZOFURANS (PCDDs & PCDFs)

The PCDDs and PCDFs present a unique and complex risk assessment problem for a number of reasons. The reader is referred to Appendix D where these issues, briefly outlined below, are treated in greater depth.

(i) The best studied PCDD isomer -- 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) -- is the most potent chemical carcinogen ever tested in laboratory animals. However certain aspects of its behavior differ from other strong carcinogens. For example, it is not mutagenic in current assays. See Appendix D, Section A, Evidence for the Carcinogenicity of PCDD/PCDF.

(ii) The PCDDs/PCDFs are present as a complex mixture, from mono-chloro through octa-chloro species. All have different toxic and carcinogenic potency. See Appendix D, section B, Acute Toxicity and Scaling Factors.

(iii) PCDDs/PCDFs are bioavailable to varying degrees depending on route of exposure and physical form (whether in vapor phase or tenaciously adsorbed to fly ash). See Appendix D, Section C, Ingestion of Contaminated Soil; Section D, Inhalation; and section E, Dermal Contact and Absorption.

(iv) The characteristics of the mechanism of action of the PCDDs/PCDFs are dissimilar to those often observed for strong carcinogens. See Appendix D, Section F, Mechanisms and Biological Implications.

In the risk assessment of the PCDDs and PCDFs, we adopt the methodology used in the F.C. Hart report. However, we use the

more recently available information on the factors mentioned above to better estimate the potential risk. In addition, we have incorporated several suggestions by the expert review committee appointed by the Board of Health and Hospitals in our methodology.

The PCDDs and PCDFs have been demonstrated to be highly carcinogenic in animal experiments, and persistent in the environment. Therefore, the risk assessment considers three routes of exposure: inhalation, ingestion of particulate-bound material in soil, and dermal absorption of material in soil and dust. In performing the risk assessment for projected levels of PCDDs and PCDFs, several assumptions must be clarified. First, since the carcinogenic potency of the various congeners and isomers vary, the relative concentrations were prorated for carcinogenic potency relative to the most carcinogenic and well characterized species, 2,3,7,8-tetrachlorodibenzo(p)dioxin (2,3,7,8-TCDD) according to current (1985) EPA recommendations. The compounds and scaling factors appear in the table on page 50. Since the mono- through tri-chloro and octa-chloro species are assigned a toxicity scaling factor of zero, they will not be included in the risk assessment as is standard procedure. The scaling factors on page 50 differ from those used in the Hart report to reflect the more recent EPA recommendations accounting more completely for relative toxicities.

EPA 2,3,7,8-TCDD Toxicity Equivalence Scaling Factors

Compound	Scaling Factor
Mono, di, and tri CDDs	0
2,3,7,8-TCDD	1
other Tetra-CDDs	0.01
2,3,7,8-PeCDDs	0.5
other Penta-CDDs	0.005
2,3,7,8-HxCDDs	0.04
other Hexa-CDDs	0.0004
2,3,7,8-HpCDDs	0.001
other Hepta-CDDs	0.00001
Octa-CDD	0
Mono, di, and tri CDFs	0
2,3,7,8-TCDFs	0.1
other Tetra-CDFs	0.001
2,3,7,8-PeCDFs	0.1
other Penta-CDFs	0.001
2,3,7,8-HxCDFs	0.01
other Hexa-CDFs	0.0001
2,3,7,8-HpCDFs	0.0001
other Hepta-CDFs	0.00001
Octa-CDF	0

All PCDD and PCDF concentrations and exposures are converted to and expressed as 2,3,7,8-TCDD equivalents as is the usual risk assessment procedure. The following table presents the 2,3,7,8-TCDD equivalents in air and the weighted deposition rate of 2,3,7,8-TCDD equivalents applied to particulate deposition.

Total 2,3,7,8 TCDD Equivalent Concentrations in Air and Deposition Rates

		Weighted Concentration		Weighted Deposition Rate	
		Air (ug/m^3)		Soil (ug/gm/day)	
Pollutants	2378-TCDD TEF[1]	Case I[2]	Case II[3]	Case I[2]	Case II[3]
PCDD/PCDF (vapor phase)		2.1E-06	2.2E-07	-	-
2378-TCDD	1	1.6E-09	1.6E-10	3.6E-11	3.8E-12
Other TCDD	0.01	2.2E-10	2.3E-11	5.0E-12	5.1E-13
2378-PeCDD	0.5	5.6E-09	5.7E-10	1.3E-10	1.3E-11
Other PeCDD	0.005	1.6E-10	1.6E-11	3.6E-12	3.7E-13
2378-HxCDD	0.04	3.1E-10	3.2E-11	7.0E-12	7.2E-13
Other HxCDD	0.004	2.2E-10	2.3E-11	5.0E-12	5.1E-13
2378-HpCDD	0.001	1.5E-11	1.5E-12	3.4E-13	3.5E-14
Other HpCDD	0.00001	1.5E-13	1.5E-14	3.4E-13	3.5E-16
2378-TCDF	0.1	2.6E-09	2.6E-10	5.8E-11	6.0E-12
Other TCDF	0.001	3.3E-10	3.4E-11	7.5E-12	7.7E-13
2378-PeCDF	0.1	3.8E-09	3.9E-10	8.5E-11	8.9E-12
Other PeCDF	0.001	6.7E-11	6.9E-12	1.5E-12	1.6E-13
2378-HxCDF	0.01	2.6E-10	2.7E-11	6.0E-12	6.2E-13
Other HxCDF	0.0001	2.2E-11	2.2E-12	4.9E-13	5.1E-14
2378-HpCDF	0.0001	1.5E-12	1.5E-13	3.3E-14	3.4E-15
Other HpCDF	0.00001	1.2E-13	1.5E-14	3.3E-15	3.4E-16
Total 2378-TCDD Equivalents		1.5E-08	1.5E-09	--	--

[1] US EPA 2,3,7,8 TCDD Toxicity Equivalence Factors.

[2] Worst Case calculated as X/Q (s/m^3) multiplied by emission rate (g/s), where X/Q is based on a normalized emission rate (1.0 g/s) for receptor location distance 0.9km, 110 degrees.

[3] Average calculated as X/Q (s/m^3) multiplied by emission rate (g/s), where X/Q is the average of X/Q for all receptors.

In addition to the assumptions made previously about air inhalation, deposition, soil and dust ingestion and dermal contact, several worst case assumptions were made in the carcinogenic risk assessment of the PCDDs/PCDFs:

- it was assumed that all airborne PCDD/PCDF is in the vapor phase and that 100% is bioavailable via inhalation. More likely than not, the majority of 2,3,7,8-TCDD equivalents are emitted adsorbed to particulates and air concentrations are cut down drastically by the Electrostatic Precipitator (ESP).

- 2,3,7,8-TCDD has been shown to be tenaciously adsorbed to particulates, especially flyash. It was assumed that 10% and 1% is bioavailable via ingestion and dermal absorption respectively. See Appendix D, Section C and E.

- For risk assessment of deposited PCDD/PCDF, we assumed that 100% is present in emissions adsorbed to particulate matter, and that it all deposits in soil and dust.

- our steady state deposition model assumes that it all deposited 2,3,7,8-TCDD remains in the soil. Clearly, with runoff, volatilization, UV degradation, etc., this is an overestimate.

Given these very worst case assumptions, the excess risk of cancer attributable to the 2,3,7,8-TCDD content projected for Facility emissions was calculated using the EPA/CAG carcinogenic potency slope of $1.56 \times 10^{+5}$ $(mg/kg/day)^{-1}$. Because EPA/CAG potency slopes are based on daily intake in miligrams per kilogram of body weight, the expected lifetime daily intake of

2,3,7,8-TCDD from all routes of exposure is expressed accordingly. See the table on the following page.

It can be seen from the values in the table that, even with the worst case assumptions made, the excess risk of cancer is far less than one in a million, and for the average exposure scenario case it is less than one in ten million.

CHLOROBENZENES

The level of chlorobenzenes in the air is expected to be several hundred times lower than those required for chronic toxic health effects. Of the total chlorobenzenes projected in Facility emissions, only hexachlorobenzene is known to be carcinogenic (IARC, 1982). The tables below provide air guidance and average daily intake information for the chlorobenzenes and hexachlorobenzenes.

Chlorobenzene: Limits in Air
ACGIH, 350 mg/m^3
Federal Standard, 350 mg/m^3

No Standards for Hexachlorobenzene

Hexachlorobenzene: In the Diet*
6.0 - 41.0 ug/kg in food, 30 US cities
397.8 ug - average daily intake in US, 1973
72.5 ug - average daily intake in the US, 1974

* IARC, 1979

A carcinogenic risk assessment was performed for hexachlorobenzene alone using the EPA/CAG potency factor for hexachlorobenzene. Since hexachlorobenzene is very persistent in the environment, ingestion in soil and dermal absorption was included with inhalation as routes of exposure.

Lifetime Daily Intake (mg/kg/day) of 2,3,7,8-TCDD Equivalents from all Routes of Exposure and Estimated Excess Lifetime Cancer Risk

	Inhaled (mg/kg/day)	Ingested In Soil (mg/kg/day)	Dermal Absorption (mg/kg/day)	Total (mg/kg/day)	EPA Carcinogenic Potency Slope $(mg/kg/day)^{-1}$	Lifetime Excess Cancer Risk
Case I	5.4×10^{-12}	5.5×10^{-14}	4.2×10^{-15}	5.4×10^{-12}	1.6×10^{5}	8.5×10^{-7}
Case II	5.5×10^{-13}	5.6×10^{-15}	4.3×10^{-16}	5.6×10^{-13}	1.6×10^{5}	8.7×10^{-8}

Assuming 100% availability from air intake, 10% availability from ingested soil, 1% available from dermal exposure.

Animal experiments have demonstrated that hexachlorobenzene is readily absorbed from the gastrointestinal tract and excreted slowly (IARC, 1979). For risk assessment purposes, it was assumed that 100% of inhaled and ingested hexachlorobenzene is bioavailable. It was also assumed that hexachlorobenzene is bioavailable via dermal absorption at 1%, similar to 2,3,7,8-TCDD.

The following table shows the lifetime daily dose of hexachlorobenzene from all routes of exposure and the calculated excess lifetime risk of cancer.

It can be seen from the table, that the worst case and realistic estimates of excess lifetime cancer risk due to hexachlorobenzene in Facility emissions are vanishingly small.

CHLOROPHENOLS

The chlorinated phenols are a group of 19 isomers of phenol with substituted chlorines. Pentachlorophenol and some tetrachlorophenols are used worldwide as wood preservatives and fungicides. Residues of the chlorophenols have been found in soil, air, water, food and human tissue samples. Sources of environmental contamination with the chlorophenols are industrial effluents, agricultural runoff, breakdown of chlorophenoxyacetic herbicides and spontaneous formation during the chlorination of drinking water for disinfection and deodorization. Acute toxicity is relatively low and the chlorophenols are not known to be mutagens or teratogens (Exxon, 1984).

Lifetime Daily Intake (mg/kg/day) of Hexachlorobenzene from all Routes of Exposure and Estimated Excess Lifetime Cancer Risk

	Air (mg/kg/day)	Ingested in Soil (mg/kg/day)	Dermal Absorption (mg/kg/day)	Total (mg/kg/day)	EPA Carcinogenic Potency Slope $(mg/kg/day)^{-1}$	Lifetime Excess Cancer Risk
Case I	2.0×10^{-10}	2.0×10^{-11}	1.5×10^{-13}	2.2×10^{-10}	1.67	3.6×10^{-10}
Case II	2.0×10^{-11}	2.1×10^{-12}	1.6×10^{-14}	2.2×11^{-13}	1.67	3.7×10^{-11}

Assuming 100% availability from air intake and soil ingestion and 1% dermal exposure.

The following information was summarized from IARC (1979); Exxon (1984), and Sittig (1985).

TOXICITY AND OCCURRENCE

PENTACHLOROPHENOL:
minimum human lethal dose, 29,000 ug/kg
NOAEL, rats, 3,000-10,000 ug/kg/day
daily intake in food, 0.76 ug/day
mean urine levels in general US population, 6.3 ug/l

REGULATORY INFORMATION

TRICHLOROPHENOL:
no standards set

PENTACHLOROPHENOL:
ACGIH TWA (8 hour) 500 ug/m^3 in air
NRC NOAEL in drinking water, 20 ug/l
EPA, safe levels in drinking water, based on toxicity data, 1,010 ug/l
EPA, safe levels in drinking water, based on taste tests, 30 ug/l

Of all the chlorophenols, two are toxicologically important; 2,4,6-trichlorophenol and pentachlorophenol. As the health effects endpoints used to evaluate these two chemicals are different, they are discussed separately.

2,4,6-TRICHLOROPHENOL

2,4,6-trichlorophenol is a weak carcinogen (Exon,1984). It is classified as an animal carcinogen although information on human carcinogenesis is incomplete (IARC, 1979). In lieu of carcinogenicity information on the rest of the trichlorophenols, it was conservatively assumed that all have the same carcinogenic potency as the 2,4,6-isomer. A carcinogenic risk assessment was

performed for the trichlorophenols projected in emissions using the EPA/CAG unit risk factor developed for 2,4,6-trichlorophenol. It is not known to be persistent in the environment, therefore, only inhalation was considered in the risk assessment. It was assumed that 100% of inhaled trichlorophenol is bioavailable. Tabulated below are the lifetime daily intake of trichlorophenol projected from inhalation and the calculated excess lifetime risk of cancer.

Lifetime Daily Intake (mg/kg/day) of Trichlorophenol and Calculated Excess Lifetime Risk of Cancer

	Daily Intake (mg/kg/day)	EPA/CAG Carcinogenic Potency Slope $(mg/kg/day)^{-1}$	Excess Lifetime Cancer Risk
Case I	1.7×10^{-9}	2.0×10^{-2}	3.4×10^{-11}
Case II	1.8×10^{-10}	2.0×10^{-2}	3.6×10^{-12}

It can be seen from the table that the calculated excess lifetime risk of cancer from trichlorophenol in Facility emissions is very small.

PENTACHLOROPHENOL

In experimental animals pentachlorophenol has been shown to be embryotoxic but not teratogenic at dose levels of up to 500 ppm (Exon,1984). Although these levels are many orders of magnitude higher than projected for Facility emissions, pentachlorophenol was evaluated on the basis of its fetotoxicity. A safe threshold for the fetotoxic effect of pentachlorophenol is derived based on the conservative NRC NOAEL in drinking water, 20 ug/l. The standard daily consumption of water for an adult was assumed to be 2 liters per day. Assuming 100% bioavailability, it was proposed that the safe threshold daily dose of pentachlorophenol or acceptable daily intake (ADI) is 40 ug/day.

Since pentachlorophenol is fairly persistent in the environment, exposure via soil ingestion and dermal absorption was also considered (Menzer et al, 1980). In lieu of bioavailability information, it was conservatively assumed that 100% of all inhaled and ingested pentachlorophenol, and 1% of dermally contacted pentachlorophenol is bioavailable. Risk is expressed as % of the no observable adverse effects (NOAEL) threshold which is the assumed ADI. The table below presents the results of the evaluation.

Lifetime Daily Intake (ug/day) of Pentachlorophenol from all Routes of Exposure and % ADI

	Intake from Air	Soil Ingestion	Dermal Absorption	Total Daily Intake	ADI	% ADI
Case I	3.6×10^{-7}	3.8×10^{-8}	2.9×10^{-10}	4.0×10^{-7}	40	1.0×10^{-6}
Case II	3.9×10^{-8}	3.9×10^{-9}	3.0×10^{-11}	4.3×10^{-8}	40	1.0×10^{-7}

From the worst case estimate summarized in the table above, it can be seen that the amount of pentachlorophenol projected in emissions represents about one millionth of a percent of the assumed ADI.

POLYCHLORINATED BIPHENYLS

Acute toxic effects of PCB exposure occur at levels which are many orders of magnitude higher than emissions from Facility are expected to produce. Therefore, chronic toxicity effects other than cancer was not included in the risk assessment. The PCBs are very persistent in the environment as they are resistant to biodegradation (Menzer et al, 1980). The following data on occurrence are summarized from IARC (1978) and regulatory information from The Handbook on Toxic and Hazardous Chemicals and Carcinogens (Sittig,1985).

PCBs OCCURRENCE:
suburban air; Florida, Colorado, Mississippi; 0.1 ug/m^3
lake Michigan water, 0.01 ug/l
levels in human milk, various areas in the US; 0.4-10.6 ug/l
average intake in US diet (1975), 8.7 ug/day

PCBs AIR GUIDELINES:
ACGIH 500-1000 ug/m^3
NIOSH, 10 hour TWA, 1.0 ug/m^3.

There is experimental evidence of a carcinogenic effect of some PCBs in rodents, although corresponding information for humans is inconclusive (IARC, 1978). Levels of PCBs in the diet necessary to produce liver lesions in rats and mice range as high as 0.1-1.0 gram per kg of diet, depending on the PCB chlorination content. PCBs are suspect human carcinogens and will be evaluated for cancer risk using the EPA/CAG potency factor (EPA, 1985).

The risk assessment considered three routes of exposure: inhalation, ingestion of deposited particulate-bound material in soil and dust, and dermal absorption of material in soil and dust. Animal experiments have demonstrated that high proportions of inhaled or ingested PCBs are absorbed (IARC, 1978). For risk assessment purposes, it was assumed that 100% of inhaled and ingested PCBs are bioavailable, and that, like 2,3,7,8-TCDD 1% is available via dermal absorption.

Total daily intakes of PCBs were calculated for all routes of exposure as described above and converted to mg PCBs/kg bw/day for calculating the excess risk of cancer. The results appear in the table below.

Lifetime Daily Intake (mg/kg/day) of PCBs from all Routes and Estimated Excess Lifetime Cancer Risk

	Air Intake (mg/kg/day)	Ingested In Soil (mg/kg/day)	Dermal Absorption (mg/kg/day)	Total Daily Dose (mg/kg/day)	EPA Carcinogenic Potency Slope $(mg/kg/day)^{-1}$	Lifetime Excess Cancer Risk
Case I	6.0×10^{-11}	6.1×10^{-12}	4.7×10^{-14}	6.7×10^{-11}	4.34	2.9×10^{-10}
Case II	6.1×10^{-12}	6.3×10^{-13}	4.8×10^{-15}	6.7×11^{-12}	4.34	2.9×10^{-11}

Assuming 100% availability from soil ingestion and air intake, 1% from dermal exposure.

It can be seen that the lifetime excess cancer risks attributable to the PCB content projected for Facility emissions are very low; less than three in ten billion at worst.

C. CUMULATIVE CONTRIBUTIONS OF THE HEAVY METALS TO NEPHROPATHY

The kidney is one of the most susceptible organs to the toxic effects of heavy metals. In this section the ability to damage the kidney or nephrotoxicity of the metals is discussed for those metals known to be nephrotoxic at chronic, low level exposures expected from Facility emissions are cadmium, lead and mercury.

Although the metals all affect the same organ, they affect different portions of the kidney and different functions are compromised. Some metals are far more toxic to the kidney (cadmium) at lower levels than others (lead). Therefore, the nephrotoxic potential of each metal is expressed as the percent of its individual nephrotoxic threshold concentration, e.g., the exposure necessary for the induction of detectable renal dysfunction. To assess the additive nephrotoxic potential of the heavy metals projected in Facility emissions, the cumulative effect is expressed as the sum of the percents of the respective thresholds.

CADMIUM

Cadmium does not appear to accumulate in the environment to an appreciable degree (Winter, 1982), therefore, only the risk of nephrotoxicity due to inhaled cadmium is discussed.

The kidney is considered the critical organ in cadmium poisoning as it accumulates cadmium over time. It is estimated that under normal conditions in an unpolluted environment, human kidneys have accumulated 50 ug of cadmium per gram wet weight by

the age of 50 years (Winter, 1982). Cadmium concentration in the renal cortex between 200-300 ug/g generally result in renal dysfunction. The 200 ug/g level is the most widely accepted threshold at which the first signs of renal dysfunction, i.e., beta-2-microproteinuria, occur (Ryan et al, 1982; Winter, 1982). It has been estimated that after 10 years of occupational exposure to 25 ug/m^3 of cadmium in air, the kidney may have accumulated the critical concentration of 200 ug/g (Friberg et al, 1974; Kjellstrom et al, 1977).

Assuming all the cadmium inhaled is retained and available, this 8 hour day, 5 day per week, 50 week per year, 10 year exposure adjusts to a daily lifetime exposure of cadmium in air at a concentration of 0.82 ug/m^3. It is assumed that this is the threshold of cadmium in air inhaled for a lifetime, above which the first detectable signs of compromised renal function appear.

Tabulated below are the cadmium concentration in air for the worst and average case scenarios, and the respective percents of the above nephrotoxicity threshold.

PROJECTED AIR CONCENTRATIONS OF CADMIUM AND CORRESPONDING PERCENT OF NEPHROTOXICITY THRESHOLD

	Projected Air Cd	Threshold Air Conc.	% Threshold
Worst Case	3.4×10^{-4}	0.82	4×10^{-2}
Average	3.5×10^{-5}	0.82	4×10^{-3}

It can be seen from the above table that the worst and average exposure scenarios would produce a lifetime nephrotoxic dose of cadmium of four hundredths and four thousandths of a percent respectively of the proposed threshold.

LEAD

Acute lead intoxication has been associated with varying degrees of renal damage. Most frequently, reports of diminution of renal function associated with lead exposure come from clinical case histories of workers exposed to such massive amounts of lead as to land in the hospital at times in their histories with neuromuscular effects and "lead colic" episodes (Cramer, et al, 1974; Lilis, et al, 1979; Radosevic, et al, 1961). These workers, employed in occupations as ship wreckers, smelters, etc., were regularly exposed to lead in air levels in excess of 200 ug/m^3 (Cramer, et al, 1974).

The decrement in renal function, i.e., in glomerular filtration rate, in lead exposed individuals, has been expressed as a concomitant rise in serum creatinine and blood urea nitrogen (BUN) (Lilis et al, 1979). Mean BUN and creatinine levels were significantly higher in subjects with higher blood lead levels, i.e., greater than 600 ug/l (Lilis, et al, 1979). Campbell et al (1977) studied individuals chronically exposed to lead in drinking water, at concentrations of greater than 100 ug/l for a mean duration of 21.5 years. It was reported that, of the 283 subjects, 57 had elevated serum urea, and of this group, 13 had blood lead levels greater than 410 ug/l versus 5 control subjects with normal renal function. There was no evidence of clinically apparent disease in any of the subjects. Therefore, it is assumed that, although blood lead levels of 600-700 ug/l have been associated with reversible renal dysfunction (IARC, 1980), a blood lead level of 410 ug/l is the threshold at which the first signs of compromised renal function appear. The table below presents the total air and soil lead contribution of Facility emissions to blood lead levels, and the respective percent of nephrotoxic threshold for the worst case and average scenario. At worst, total lead in emissions provide 0.05% of the threshold blood level, and more likely, 5.7×10^{-3}%.

Lead Emissions

and Estimated Corresponding Blood Lead Levels

	Total Contribution to blood lead ug/l	Nephrotoxicity Threshold	% Nephrotoxicity Threshold
Worst Case	0.21	410	0.05
Average	0.023	410	5.7×10^{-3}

MERCURY

The nephrotoxic effects of chronic exposure to mercury were discussed in the section on mercury in the section addressing the regulated pollutants. The worst case and average contribution to the nephrotoxic mercury blood level are 2×10^{-3}, and 2×10^{-4}% respectively (see table on page 22).

CUMULATIVE NEPHROTOXICITY

The individual nephrotoxic contributions of cadmium, lead and mercury are tabulated in below.

Total Heavy Metal Contribution to Nephrotoxic Threshold

	CADMIUM	LEAD	MERCURY	TOTAL %
Worst Case	4.0×10^{-2}	5.0×10^{-2}	2.0×10^{-3}	9.2×10^{-2}
Average	4.0×10^{-3}	5.7×10^{-3}	2.0×10^{-4}	9.9×10^{-3}

It can be seen from the last column in the above table that the maximum heavy metal contribution to nephrotoxicity is on the level of hundredths of a percent, and more realistically, thousandths of one percent of the estimated thresholds.

V. RESULTS AND DISCUSSION

The estimates for the excess lifetime risk of cancer attributable to exposure to the various pollutants are summarized below. The bottom line of the table gives the overall cancer risk estimate for both Case I, the worst case scenario, and Case II, a more realistic average scenario. In addition, the respective and additive percentages which pollutants contribute to other health effects thresholds, e.g., nephrotoxicity and fetotoxicity, are summarized in the table on the following page as above.

Summary of Excess Lifetime Risk of
Cancer Estimated for Facility Emissions

	Cancer Risk	
Pollutant	Case I	Case II
Arsenic	3.8×10^{-7}	3.9×10^{-8}
Cadmium	3.7×10^{-7}	3.8×10^{-8}
Chromium	1.2×10^{-7}	1.2×10^{-8}
Nickel	3.4×10^{-8}	3.5×10^{-9}
Formaldehyde	7.2×10^{-8}	6.5×10^{-9}
Carcinogenic PAH	1.1×10^{-9}	1.1×10^{-10}
PCDD/PCDF	8.5×10^{-7}	8.7×10^{-8}
Hexachlorobenzene	3.6×10^{-10}	3.7×10^{-11}
Trichlorophenol	3.4×10^{-11}	3.6×10^{-12}
PCB	2.9×10^{-10}	2.9×10^{-11}
Total	1.8×10^{-6}	1.9×10^{-7}

Estimated Percent Contribution to the Nephrotoxicity Threshold for the Heavy Metals

Metal	% Threshold Case I	% Threshold Case II
Cadmium	4.0×10^{-2}	4.0×10^{-3}
Lead	5.0×10^{-2}	5.7×10^{-3}
Mercury	2.0×10^{-3}	2.0×10^{-4}
Total	9.2×10^{-2}	9.9×10^{-3}

Estimated Percent Contribution to Fetotoxicity Threshold For Pentachlorophenol

	% Threshold Case I	% Threshold Case II
Pentachlorophenol	1.0×10^{-6}	1.0×10^{-7}

The overall individual risk of developing cancer if exposed to pollutants from the facility over the course of a lifetime is estimated to be 1.8×10^{-6} and 1.9×10^{-7} (or less than two in a million and less than two in ten million) for Case I and Case II respectively.

It must be kept in mind that these risk estimates represent the upper-bound risk on three levels. First, on the level of the exposure analyses, the concentrations and deposition rates are calculated from maximum projected emission data.

Second, the assumptions used in estimating intake almost certainly overestimate the actual quantity the individual is

exposed to. This uncertainty is inherent in the estimates we use for soil ingestion, adherence to skin, and the estimates in bioavailability from all routes of exposure.

Third, the EPA/CAG unit risk for an air pollutant is defined as the excess lifetime cancer risk occurring in a hypothetical population in which all individuals are exposed continuously from birth throughout their lifetimes to a concentration of 1 part per million or 1 ug/m^3 of the agent in the air they breathe. There are many reasons why this factor has some built-in and probably very large uncertainties. For one thing, ours is a mobile society, the likelihood of anyone spending an entire lifetime in one place, day in and day out is as unlikely as it sounds. Another aspect of uncertainty is inherent in the data and methodology used in developing the unit risk factors:

> The data used for quantitative estimation are taken from one or both of the following: 1) lifetime animal studies, and 2) human studies where excess cancer risk has been associated with exposure to the agent. In animal studies it is assumed, unless evidence exists to the contrary, that if a carcinogenic response occurs at the dose levels used in the study, then responses will also occur at all lower doses, with an incidence determined by an extrapolation model.
>
> There is no solid scientific basis for any mathematical extrapolation model that relates carcinogen exposure to cancer risks at the extremely low concentrations that must be dealt with in evaluating environmental hazards. For practical reasons, such low levels of risk cannot be measured directly either by animal experiments or by epidemiologic studies.

> The linear nonthreshold model has been adopted as the primary basis for risk extrapolation to low levels of the dose-response relationship. The incremental risk estimates made with this model, and the corresponding 95% upper-limit incremental unit risks, should be regarded as conservative, representing the most plausible upper limits for the risk, i.e., the true risk is not likely to be higher than the estimates, but it could be lower.
>
> Source: EPA, 1985

The net result is a high risk estimate:

"The method used by the CAG for quantitative extrapolation from animal to man are generally conservative, i.e., tending toward high estimates of risk." (EPA, 1985)

An overall lifetime cancer risk of 1.8 in a million and 1.9 in ten million are very small numbers. They represent non-zero probabilities of developing cancer. But, how small actually are these probabilities? These risks do not occur in a vacuum but are superimposed on the risks we incur in our daily lives. To lend a real life perspective to the idea of a one in a million risk, the following table presents a number of everyday events, occurrences or occupations and the time it takes to accumulate a one in a million risk of death.

Some one in a Million Risks

Living in the United States: Time to accumulate a one in a million risk of death from the cause indicated.

Motor vehicle accident	1.5 days
Falls	6 days
Drowning	10 days
Fires	13 days
Firearms	36 days
Electrocution	2 months
Tornadoes	20 months
Floods	20 months
Lightning	2 years
Animal bite or sting	4 years

Occupational Risks. Time to accumulate a one in a million risk of death in the occupation indicated.

General	
Manufacturing	4.5 days
Trade	7 days
Service and Government	3.5 days
Transport and Public Utilities	1 day
Agriculture	15 hours
Construction	14 hours
Mining and Quarrying	9 hours

Specific	
Coal Mining (accidents)	14 hours
Police duty	1.5 days
Railroad Employment	1.5 days
Fire Fighting	11 hours

Other Risks:

Cosmic Rays

One transcontinental round trip by air.
Living 1.5 months in Colorado compared to New York.
Camping at 15,000 feet for 6 days compared to sea level.

Other Radiation

20 days of sea level natural background radiation
2.5 months in masonry rather than wood building.
1/7 of a chest X-ray using modern equipment.

Other Risks (continued):

Eating and Drinking

40 diet sodas (saccharin).
6 pounds of peanut butter (aflatoxin).
180 pints of milk (aflatoxin).
200 gallons of drinking water from Miami or New Orleans.
90 pounds of broiled steak (cancer risk only).

Smoking

2 cigarettes

Source: Wilson, et al, 1982

It must be kept in mind that the cancer risks estimated for exposure to pollutants from the Facility emissions are not the only cancer risks we face. The American Cancer Society, in its annual Cancer Statistics report estimates that one third of all Americans born this year will develop invasive cancer during their lifetime, and almost one fourth will die of it (American Cancer Society, 1985). Shown in the following table are the lifetime risks of death from the various types of cancer.

Lifetime Cancer Risks[a]

Current Cancer Rates[b]	Lifetime Average Risk[c]
All Cancers	2.0×10^{-1}
Buccal cavity, pharynx, respiratory	5.0×10^{-2}
Digestive organs and peritoneum	5.2×10^{-2}
Bone, connective tissue, skin, breast	2.2×10^{-2}
Genital organs	2.2×10^{-2}
Urinary tract	8.4×10^{-3}
Other	1.9×10^{-4}
Leukemia and other blood and lymph	1.8×10^{-4}
Cosmic Ray Risks[d]	
Airline pilot (50 hours per month at 12 kilometers altitude)	2.8×10^{-3}
One transcontinental round trip by air per year	7.0×10^{-5}
Frequent airline passenger (4 hours per week flying	7.0×10^{-4}
Living in Colorado compared to New York	5.6×10^{-4}
Other Radiation Risks	
Natural background radiation (sea level)	1.4×10^{-3}
Average diagnostic medical X-rays in the United States	1.4×10^{-3}
Living in masonry building rather than wood	3.5×10^{-4}
Eating and Drinking	
One 12 1/2 ounce diet drink per day	7.0×10^{-4}
Average saccharin consumption in the United States	1.4×10^{-4}
Four tablespoons peanut butter per day[e]	5.6×10^{-4}
One pint milk per day[e]	1.4×10^{-4}
Miami or New Orleans drinking water	7.0×10^{-5}
1/2 lb. charcoal broiled steak per week (cancer risk only; heart attack and other risks additional)	2.1×10^{-7}
Alcohol, averaged over smokers and nonsmokers[f]	3.5×10^{-3}
Alcohol, light drinker (one beer per day)[f]	1.4×10^{-3}
Tobacco[g]	
Smoker, cancer only	8.4×10^{-2}
Smoker, all effects (including heart disease)	2.1×10^{-1}
Person sharing room with smoker	7.0×10^{-4}

Lifetime Caoncer Risks[a] (continued)

Air Pollution	
Polycyclic organics, all effects	1.05×10^{-3}

a. These risks of death, the difference between incidence and mortality being well within the uncertainties shown, (except for the Current Cancer Rates category).

b. Included to give some perspective. The figures given correspond approximately to the lifetime risk. The lifetime risk is estimated by the fraction of those dying who die of the given cancer, average lifetime is estimated as seventy years. Since cancer rates increase rapidly with age and the population age structure is changing, these figures are only approximate. Data from Vital Statistics of the United States, 1975.

c. Averaged over the whole population of the United States, assuming a 70 year lifetime.

d. We assume a linear model with a total of 1 cancer per 5,000 man-rem, corresponding to BEIR 1972. More recent estimates of the BEIR committee (1980) would give slightly lower estimates.

e. Based on human data for aflatoxin carcinogenicity. Note that we assume that the measured aflotoxins are aflatoxin B, the most potent. If some corresponds to other aflotoxins, these estimated risks should be reduced.

f. Cirrhosis of the liver. Not a cancer, but included here since the methods used are similar. It is possible that in this case there is a threshold effect for damage. In addition there is some evidence that moderate alcohol consumption is associated with lower death rates from other diseases.

g. Based on human data.

Source: Wilson, *et al*, 1982

What all this means is that the average individual has a lifetime risk of death from cancer of 0.25. This translates to about 244,222 cancer deaths in the lifetime of our entire estimated at-risk population of 976,888. For the method used in estimating the at-risk population see Appendix C. The *excess* lifetime cancer risk from exposure to Facility emissions is *superimposed* on this already large risk. For the individual, the

worst case risk of cancer is 1.8 in a million, and the average risk is 1.9 in ten million. For the population as a whole, this translates to an excess cancer death of about 1.76 for the worst case scenario, or 0.19 for our average risk scenario over the 70 year lifetime of the population. Basically, this means that under the worst of all possible conditions, a less than _7 ten thousandths of a percent_ increase in the cancer death rate may occur. More likely than not, an increase would be invisible for a population of this size (976,880) as the expected number of excess deaths is less than 1 (0.19) for the average risk scenario.

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APPENDIX A

Average Annual Concentration and Deposition Rate Calculation for the Air Impact Receptor Array

MEMORANDUM

TO: Susan Baer

FROM: David Bull

RE: City of Boston Waste-to-Energy Project: Average Annual Concentration and Deposition Rate Calculation for the Air Impact Receptor Array

DATE: 10 October 1986

Overview

The attached spreadsheets summarize the data and methodology used to arrive at an average annual concentration (ug/m^3) and deposition rate (gm/m^2) for the total receptor array.

Derivation

There are 10 defined receptor rings, each comprised of 36 receptor points, for a total of 360 points. Normalized average annual concentrations and deposition rates for each of the 10 receptor rings, as determined by the air impact modeling, were averaged (see spreadsheet). Next, a weighting factor was calculated for each of the 10 receptor rings and applied to the average concentration and deposition rate. The weighting factor considers the increase in the area of each receptor ring as one travels away from the stack. Specifically, the area of each receptor ring (1 through 10) was calculated and divided by the total area of the receptor array (i.e., the ratio of the area of a given receptor ring to the total receptor array area). The resulting ratio is the weighting factor applied to the averages calculated for each of the 10 receptor rings and shown in the summary table.

If you have any questions, please don't hesitate to call.

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PROJECT: BOSTON


SUMMARY : Weighted Average of Concentrations and Deposition Rates
          at the 10 Concentric Receptor Rings. Each Receptor
          Ring Contains 36 Receptor Points.

Receptor             CONCEN-
Ring                 TRATION             DEPOSITION
                     (ug/m3)              (gm/m2)
---------           ---------           ---------
        1            0.0001              0.00001
        2            0.0001              0.00002
        3            0.0002              0.00002
        4            0.0004              0.00005
        5            0.0004              0.00006
        6            0.0004              0.00005
        7            0.0004              0.00005
        8            0.0006              0.00008
        9            0.0014              0.00018
       10            0.0019              0.00032
                    ---------           ---------

AVERAGE FOR
TOTAL ARRAY   =      0.0057              0.00084

                    ---------           ---------
                    ---------           ---------
```

PROJECT: BOSTON

WEIGHTING DERIVATION TABLE

receptor r (km)	r2 (km2)	pi	area pi*r2 (km2)	receptor wt.
0.6	0.36	3.1416	1.13	0.0044
0.9	0.81	3.1416	2.54	0.0056
1.2	1.44	3.1416	4.52	0.0078
1.7	2.89	3.1416	9.07	0.0179
2.2	4.84	3.1416	15.2	0.0241
2.7	7.29	3.1416	22.9	0.0302
3.2	10.24	3.1416	32.1	0.0364
4	16	3.1416	50.2	0.0711
6	36	3.1416	113.	0.2469
9	81	3.1416	254.	0.5556
overall area			254.	1.0000

```
PROJECT: BOSTON

                                                                                                          10-Oct-86

RECEP        1             | RECEP       2             | RECEP       3             | RECEP       4             | RECEP       5
                           |                           |                           |                           |
Dist       0.6 km          | Dist      0.9 km          | Dist      1.2 km          | Dist      1.7 km          | Dist      2.2 km
---------------------------|---------------------------|---------------------------|---------------------------|---------------------------
Recepto    CON.     DEP.   | Recep    CON.     DEP.    | Recep    CON.     DEP.    | Recep    CON.     DEP.    | Recep    CON.     DEP.
   #      (avg)    (avg)   |   #     (avg)    (avg)    |   #     (avg)    (avg)    |   #     (avg)    (avg)    |   #     (avg)    (avg)
 -----    -----    -----   | -----   -----    -----    | -----   -----    -----    | -----   -----    -----    | -----   -----    -----
     1   0.0142  0.00188   |     2  0.0224  0.00296    |     3  0.0230  0.00305    |     4  0.0186  0.00247    |     5  0.0160  0.00211
    11   0.0153  0.00203   |    12  0.0254  0.00337    |    13  0.0258  0.00342    |    14  0.0212  0.00281    |    15  0.0173  0.00229
    21   0.0142  0.00188   |    22  0.0231  0.00306    |    23  0.0229  0.00304    |    24  0.0182  0.00242    |    25  0.0142  0.00188
    31   0.0162  0.00215   |    32  0.0265  0.00351    |    33  0.0257  0.00340    |    34  0.0204  0.00270    |    35  0.0161  0.00214
    41   0.0212  0.00281   |    42  0.0344  0.00456    |    43  0.0344  0.00456    |    44  0.0268  0.00355    |    45  0.0215  0.00285
    51   0.0235  0.00311   |    52  0.0374  0.00496    |    53  0.0372  0.00493    |    54  0.0291  0.00385    |    55  0.0234  0.00310
    61   0.0208  0.00276   |    62  0.0318  0.00421    |    63  0.0307  0.00407    |    64  0.0237  0.00313    |    65  0.0181  0.00240
    71   0.0197  0.00260   |    72  0.0286  0.00379    |    73  0.0274  0.00363    |    74  0.0203  0.00269    |    75  0.0144  0.00191
    81   0.0245  0.00324   |    82  0.0360  0.00477    |    83  0.0349  0.00462    |    84  0.0283  0.00375    |    85  0.0212  0.00281
    91   0.0294  0.00390   |    92  0.0456  0.00604    |    93  0.0430  0.00570    |    94  0.0394  0.00523    |    95  0.0277  0.00367
   101   0.0307  0.00407   |   102  0.0484  0.00641    |   103  0.0450  0.00595    |   104  0.0353  0.00468    |   105  0.0267  0.00353
   111   0.0286  0.00379   |   112  0.0457  0.00605    |   113  0.0428  0.00567    |   114  0.0328  0.00434    |   115  0.0255  0.00338
   121   0.0258  0.00342   |   122  0.0388  0.00514    |   123  0.0355  0.00470    |   124  0.0277  0.00366    |   125  0.0217  0.00287
   131   0.0199  0.00263   |   132  0.0331  0.00439    |   133  0.0317  0.00420    |   134  0.0252  0.00334    |   135  0.0198  0.00263
   141   0.0154  0.00204   |   142  0.0255  0.00338    |   143  0.0250  0.00332    |   144  0.0210  0.00278    |   145  0.0163  0.00216
   151   0.0124  0.00165   |   152  0.0220  0.00291    |   153  0.0222  0.00294    |   154  0.0188  0.00249    |   155  0.0152  0.00202
   161   0.0116  0.00153   |   162  0.0216  0.00286    |   163  0.0223  0.00295    |   164  0.0194  0.00257    |   165  0.0185  0.00245
   171   0.0115  0.00153   |   172  0.0221  0.00292    |   173  0.0233  0.00308    |   174  0.0212  0.00281    |   175  0.0193  0.00256
   181   0.0094  0.00124   |   182  0.0180  0.00238    |   183  0.0200  0.00265    |   184  0.0191  0.00253    |   185  0.0154  0.00204
   191   0.0073  0.00097   |   192  0.0144  0.00191    |   193  0.0154  0.00204    |   194  0.0146  0.00194    |   195  0.0122  0.00162
   201   0.0071  0.00094   |   202  0.0134  0.00178    |   203  0.0148  0.00196    |   204  0.0127  0.00168    |   205  0.0102  0.00135
   211   0.0082  0.00109   |   212  0.0152  0.00201    |   213  0.0170  0.00226    |   214  0.0144  0.00190    |   215  0.0116  0.00154
   221   0.0088  0.00116   |   222  0.0160  0.00212    |   223  0.0178  0.00236    |   224  0.0151  0.00200    |   225  0.0114  0.00151
   231   0.0090  0.00119   |   232  0.0163  0.00216    |   233  0.0170  0.00226    |   234  0.0153  0.00203    |   235  0.0119  0.00158
   241   0.0102  0.00135   |   242  0.0171  0.00226    |   243  0.0175  0.00232    |   244  0.0151  0.00200    |   245  0.0130  0.00172
   251   0.0123  0.00163   |   252  0.0194  0.00257    |   253  0.0194  0.00257    |   254  0.0162  0.00214    |   255  0.0128  0.00170
   261   0.0138  0.00183   |   262  0.0208  0.00275    |   263  0.0200  0.00264    |   264  0.0160  0.00212    |   265  0.0126  0.00167
   271   0.0143  0.00190   |   272  0.0206  0.00272    |   273  0.0192  0.00255    |   274  0.0151  0.00200    |   275  0.0113  0.00150
   281   0.0165  0.00219   |   282  0.0235  0.00311    |   283  0.0220  0.00291    |   284  0.0173  0.00230    |   285  0.0130  0.00173
   291   0.0181  0.00240   |   292  0.0257  0.00341    |   293  0.0242  0.00321    |   294  0.0191  0.00253    |   295  0.0146  0.00193
   301   0.0157  0.00208   |   302  0.0220  0.00291    |   303  0.0206  0.00273    |   304  0.0163  0.00215    |   305  0.0125  0.00165
   311   0.0114  0.00151   |   312  0.0159  0.00211    |   313  0.0150  0.00198    |   314  0.0118  0.00156    |   315  0.0093  0.00124
   321   0.0078  0.00103   |   322  0.0115  0.00153    |   323  0.0111  0.00147    |   324  0.0090  0.00119    |   325  0.0072  0.00950
   331   0.0061  0.00081   |   332  0.0103  0.00137    |   333  0.0106  0.00140    |   334  0.0090  0.00119    |   335  0.0074  0.00980
   341   0.0067  0.00089   |   342  0.0121  0.00161    |   343  0.0126  0.00167    |   344  0.0110  0.00146    |   345  0.0097  0.00129
   351   0.0100  0.00132   |   352  0.0172  0.00228    |   353  0.0175  0.00232    |   354  0.0146  0.00194    |   355  0.0129  0.00171
          -----    -----             -----    -----             -----    -----             -----    -----             -----    -----
avg.     0.0152  0.00202            0.0244  0.00323            0.0240  0.00318            0.0197  0.00261            0.0156  0.00255
wt.      0.0044   0.0044            0.0056   0.0056            0.0078   0.0078            0.0179   0.0179            0.0241   0.0241

wt.avg.  0.0001  0.00001            0.0001  0.00002            0.0002  0.00002            0.0004  0.00005            0.0004  0.00006

WEIGHTING DERIVATION TABLE
--------------------------

receptor                 area
```

PROJECT: BOSTON

10-Oct-86

```
! RECEP        6           ! RECEP        7          ! RECEP        8           ! RECEP        9           ! RECEP        10          !
!                          !                         !                           !                           !                           !
! Dist      2.7 km         ! Dist      3.2 km        ! Dist      4.0 km          ! Dist      6.0 km          ! Dist      9.0 km          !
!------------------------- !-------------------------!---------------------------!---------------------------!---------------------------!
! Recep   CON.      DEP.   ! Recep   CON.     DEP.   ! Recep   CON.      DEP.    ! Recep   CON.      DEP.    ! Recep   CON.      DEP.    !
!   #     (avg)     (avg)  !   #     (avg)    (avg)  !   #     (avg)     (avg)   !   #     (avg)     (avg)   !   #     (avg)     (avg)   !
! -----   -----     -----  ! -----   -----    -----  ! -----   -----     -----   ! -----   -----     -----   ! -----   -----     -----   !
!    6   0.0139   0.00184  !    7   0.0104  0.00137  !    8   0.0087   0.00115   !    9   0.0053   0.00070   !   10   0.0034   0.00045   !
!   16   0.0145   0.00192  !   17   0.0119  0.00157  !   18   0.0079   0.00131   !   19   0.0067   0.00088   !   20   0.0043   0.00057   !
!   26   0.0114   0.00152  !   27   0.0092  0.00122  !   28   0.0073   0.00097   !   29   0.0049   0.00064   !   30   0.0030   0.00040   !
!   36   0.0132   0.00174  !   37   0.0109  0.00144  !   38   0.0093   0.00124   !   39   0.0058   0.00077   !   40   0.0038   0.00050   !
!   46   0.0177   0.00235  !   47   0.0148  0.00195  !   48   0.0119   0.00158   !   49   0.0077   0.00102   !   50   0.0052   0.00069   !
!   56   0.0194   0.00257  !   57   0.0165  0.00219  !   58   0.0132   0.00175   !   59   0.0089   0.00118   !   60   0.0058   0.00077   !
!   66   0.0147   0.00195  !   67   0.0123  0.00163  !   68   0.0097   0.00128   !   69   0.0061   0.00081   !   70   0.0041   0.00054   !
!   76   0.0112   0.00149  !   77   0.0091  0.00121  !   78   0.0069   0.00091   !   79   0.0041   0.00054   !   80   0.0024   0.00032   !
!   86   0.0162   0.00215  !   87   0.0131  0.00174  !   88   0.0098   0.00130   !   89   0.0059   0.00078   !   90   0.0037   0.00049   !
!   96   0.0210   0.00278  !   97   0.0171  0.00226  !   98   0.0129   0.00171   !   99   0.0084   0.00112   !  100   0.0052   0.00069   !
!  106   0.0217   0.00287  !  107   0.0179  0.00237  !  108   0.0142   0.00188   !  109   0.0098   0.00129   !  110   0.0059   0.00079   !
!  116   0.0210   0.00278  !  117   0.0178  0.00236  !  118   0.0147   0.00194   !  119   0.0104   0.00138   !  120   0.0060   0.00079   !
!  126   0.0176   0.00233  !  127   0.0146  0.00194  !  128   0.0113   0.00150   !  129   0.0078   0.00104   !  130   0.0045   0.00059   !
!  136   0.0164   0.00217  !  137   0.0139  0.00184  !  138   0.0111   0.00148   !  139   0.0079   0.00105   !  140   0.0047   0.00062   !
!  146   0.0148   0.00195  !  147   0.0128  0.00170  !  148   0.0086   0.00114   !  149   0.0055   0.00073   !  150   0.0035   0.00046   !
!  156   0.0136   0.00180  !  157   0.0115  0.00152  !  158   0.0089   0.00117   !  159   0.0059   0.00079   !  160   0.0038   0.00051   !
!  166   0.0147   0.00195  !  167   0.0115  0.00153  !  168   0.0095   0.00126   !  169   0.0058   0.00077   !  170   0.0037   0.00500   !
!  176   0.0160   0.00211  !  177   0.0141  0.00187  !  178   0.0103   0.00137   !  179   0.0071   0.00094   !  180   0.0046   0.00061   !
!  186   0.0125   0.00165  !  187   0.0110  0.00146  !  188   0.0084   0.00112   !  189   0.0052   0.00069   !  190   0.0031   0.00042   !
!  196   0.0102   0.00135  !  197   0.0089  0.00118  !  198   0.0070   0.00092   !  199   0.0046   0.00061   !  200   0.0030   0.00039   !
!  206   0.0087   0.00116  !  207   0.0077  0.00102  !  208   0.0057   0.00076   !  209   0.0035   0.00046   !  210   0.0022   0.00029   !
!  216   0.0096   0.00128  !  217   0.0085  0.00113  !  218   0.0065   0.00086   !  219   0.0041   0.00054   !  220   0.0027   0.00035   !
!  226   0.0097   0.00129  !  227   0.0078  0.00103  !  228   0.0060   0.00079   !  229   0.0038   0.00050   !  230   0.0024   0.00031   !
!  236   0.0095   0.00126  !  237   0.0072  0.00096  !  238   0.0059   0.00078   !  239   0.0039   0.00051   !  240   0.0023   0.00031   !
!  246   0.0102   0.00135  !  247   0.0078  0.00104  !  248   0.0062   0.00083   !  249   0.0041   0.00054   !  250   0.0025   0.00033   !
!  256   0.0108   0.00143  !  257   0.0092  0.00121  !  258   0.0064   0.00085   !  259   0.0043   0.00057   !  260   0.0025   0.00034   !
!  266   0.0109   0.00144  !  267   0.0091  0.00120  !  268   0.0069   0.00091   !  269   0.0046   0.00061   !  270   0.0029   0.00038   !
!  276   0.0089   0.00118  !  277   0.0073  0.00096  !  278   0.0056   0.00074   !  279   0.0038   0.00050   !  280   0.0022   0.00030   !
!  286   0.0103   0.00137  !  287   0.0085  0.00113  !  288   0.0066   0.00088   !  289   0.0041   0.00055   !  290   0.0026   0.00034   !
!  296   0.0116   0.00154  !  297   0.0096  0.00127  !  298   0.0076   0.00101   !  299   0.0050   0.00066   !  300   0.0032   0.00042   !
!  306   0.0101   0.00133  !  307   0.0083  0.00110  !  308   0.0066   0.00087   !  309   0.0042   0.00056   !  310   0.0027   0.00035   !
!  316   0.0076   0.00101  !  317   0.0064  0.00085  !  318   0.0052   0.00069   !  319   0.0035   0.00046   !  320   0.0021   0.00028   !
!  326   0.0059   0.00079  !  327   0.0050  0.00066  !  328   0.0040   0.00053   !  329   0.0029   0.00038   !  330   0.0017   0.00023   !
!  336   0.0062   0.00082  !  337   0.0053  0.00071  !  338   0.0045   0.00060   !  339   0.0032   0.00043   !  340   0.0019   0.00026   !
!  346   0.0086   0.00114  !  347   0.0064  0.00085  !  348   0.0052   0.00069   !  349   0.0035   0.00047   !  350   0.0022   0.00029   !
!  356   0.0115   0.00152  !  357   0.0086  0.00114  !  358   0.0072   0.00095   !  359   0.0049   0.00065   !  360   0.0030   0.00040   !
         ------   ------           ------   ------           ------    ------            ------    ------            ------    ------
         0.0128   0.00170          0.0106  0.00141           0.0083   0.00110            0.0055   0.00073            0.0034   0.00058
         0.0302    0.0302          0.0364   0.0364           0.0711    0.0711            0.2469    0.2469            0.5556    0.5556

         0.0004   0.00005          0.0004  0.00005           0.0006   0.00008            0.0014   0.00018            0.0019   0.00032
```

APPENDIX B

Summary of Lead Test Results by Census Tract and Community in Suffolk County, Somerville, Cambridge and Brookline

Suffolk County
Cooperative Extension Service
University of Massachusetts—U.S.D.A. and Massachusetts counties cooperating

150 Causeway Street
Room 803
Boston, MA 02114
(617) 727-4107

SUMMARY OF SOIL LEAD TEST RESULTS BY CENSUS TRACT AND COMMUNITY IN SUFFOLK COUNTY, SOMERVILLE, CAMBRIDGE AND BROOKLINE

1977 - August 7, 1985

(Compiled by Michelle Doyle, Summer Assistant and Alan Schepps, Associate Extension Horticulturist-8/85)

Levels	Total Predicted Lead
Low	0-499 ppm
Medium	500-999 ppm
High	1000- 3000 ppm
Very high	Greater than 3000 ppm

Note:
- 90% or more of these samples are from gardens taken to a six inch depth, therefore diluting the highest concentrations of lead in the top half inch. Due to the present program emphasis on sampling the top half inch of play areas, future data will be summarizes by depth of sampling as well.
- Grand totals on last page.
- All individual soil test reports and information sheets are on file by census tract and year.
- For more information contact Alan Schepps at 727-4107.

Cooperative Extension offers equal opportunity in programs and employment

SUMMARY OF SOIL LEAD TEST RESULTS

1977 - August 7, 1985

CHELSEA	CLIENTS	SAMPLES	LOW	MEDIUM	HIGH	V. HIGH
1601	3	5	1	2	2	0
1602	4	9	1	1	6	1
1603	0	0	0	0	0	0
1604	15	55	21	11	16	7
1605	8	14	8	5	1	0
1606	8	22	20	2	0	0
1607	1	1	1	0	0	0
UNK.	3	14	12	1	1	0
TOTAL	42	120	64 53.3%	22 18.3%	26 21.7%	8 6.7%
WINTHROP						
1801	3	8	5	3	0	0
1802	8	10	4	6	0	0
1803	10	13	5	4	1	3
1804	4	9	7	2	0	0
1805	10	17	8	5	4	0
UNK.	7	34	15	11	5	3
TOTAL	42	91	44 48.4%	31 34.1%	10 10.9%	6 6.6%
REVERE						
1701	8	12	9	1	2	0
1702	4	11	4	7	0	0
1703	3	3	3	0	0	0
1704	1	2	2	0	0	0

REVERE	CLIENTS	SAMPLES	LOW	MEDIUM	HIGH	V. HIGH
1705	5	14	9	4	1	0
1706	7	15	7	2	6	0
1707	3	7	3	3	1	0
1708	2	3	2	1	0	0
UNK.	1	1	1	0	0	0
TOTAL	34	68	40 58.8%	18 26.5%	10 14.7%	0
BROOKLINE						
4001	9	18	12	5	1	0
4002	16	37	12	15	10	0
4003	26	41	9	14	15	3
4004	15	27	23	2	2	0
4005	25	40	24	11	5	0
4006	12	21	15	2	4	0
4007	16	42	12	20	9	1
4008	11	21	6	12	3	0
4009	15	36	10	14	12	0
4010	14	26	15	6	5	0
4011	27	95	62	21	11	1
4012	10	20	16	3	1	0
TOTAL	196	424	216 50.9%	125 29.5%	78 18.4%	5 1.2%
SOMERVILLE						
3501	6	16	4	5	6	1
3502	19	45	6	19	19	1
3503	0	0	0	0	0	0

SOMERVILLE	CLIENTS	SAMPLES	LOW	MEDIUM	HIGH	V. HIGH
3504	32	88	15	33	39	1
3505	11	31	5	12	14	0
3506	4	14	2	4	8	0
3507	13	29	16	12	0	1
3508	5	11	2	6	3	0
3509	20	39	10	11	17	1
3510	22	45	13	16	14	2
3511	22	55	19	22	14	0
3512	36	116	19	37	35	25
3513	27	70	31	20	15	4
3514	21	46	9	16	20	1
3515	6	11	2	3	6	0
UNK.	3	25	0	3	11	11
TOTAL	247	641	153 23.9%	219 34.2%	221 34.5%	48 7.7%

CAMBRIDGE						
3521	0	0	0	0	0	0
3522	1	4	4	0	0	0
3523	1	5	0	0	4	1
3524	2	6	4	0	2	0
3525	9	23	3	8	12	0
3526	1	3	0	3	0	0
527	1	1	0	1	0	0
3528	3	14	0	2	10	2
529	18	49	6	17	26	0
3530	10	26	13	0	1	2
531	1	5	0	0	5	0

CAMBRIDGE	CLIENTS	SAMPLES	LOW	MEDIUM	HIGH	V. HIGH
3532	12	44	9	13	20	2
3533	23	70	2	19	41	8
3534	10	41	8	13	20	0
3535	17	60	6	22	30	2
3536	18	65	16	28	13	8
3537	11	30	9	11	10	0
3538	9	28	12	4	12	0
3539	7	29	6	8	13	2
3540	8	25	5	10	9	1
3541	3	6	1	3	2	0
3542	19	51	35	10	5	1
3543	6	18	1	11	0	6
3544	9	27	17	7	3	0
3545	14	35	15	8	7	5
3546	10	39	17	13	8	1
3547	8	16	1	13	2	0
3548	9	26	11	10	5	0
3549	7	21	7	12	2	0
3550	7	16	0	4	10	2
3558	0	0	0	0	0	0
UNK.	8	18	7	2	7	2
TOTAL	262	801	215 26.8%	252 31.5%	289 36%	45 5.6%

BACK BAY

	CLIENTS	SAMPLES	LOW	MEDIUM	HIGH	V. HIGH
0101.01	3	11	10	1	0	0
0101.02	0	0	0	0	0	0
0102	18	56	38	10	8	0
0103	1	4	4	0	0	0
0104	5	13	11	1	1	0
0105	7	12	4	6	2	0
0106	4	9	8	1	0	0
0107	10	13	8	5	0	0
0108	7	12	8	3	1	0
UNK.	12	20	15	2	2	1
TOTAL	67	150	106 70.7%	29 19.3%	14 9.3%	1 .7%

ALLSTON-BRIGHTON

0001	27	91	50	23	16	2
0002.01	17	44	32	5	7	0
0002.02	13	26	20	4	2	0
0003	23	39	27	10	2	0
0004	32	65	41	22	2	0
0005	18	29	24	5	0	0
0006.01	13	20	7	11	2	0
0006.02	5	22	14	5	3	0
0007.01	2	5	1	4	0	0
0007.02	2	3	0	1	2	0
0008	28	72	40	23	9	0
TOTAL	180	416	256 61.5%	113 27.2%	45 10.8%	2 .5%

CHARLESTOWN

	CLIENTS	SAMPLES	LOW	MEDIUM	HIGH	V. HIGH
0401	20	46	25	11	10	0
0402	23	46	30	11	5	0
0403	5	7	3	1	3	0
0404	5	7	7	0	0	0
0405	1	1	1	0	0	0
0406	2	6	6	0	0	0
0407	18	31	18	9	3	1
0408	21	28	25	3	0	0
TOTAL	95	172	115 66.9%	35 20.3%	21 12.2%	1 .6%

NORTH DORCHESTER

0901	2	11	8	2	1	0
0902	0	0	0	0	0	0
0903	1	8	7	1	0	0
0904	4	15	0	1	13	1
0905	1	3	2	1	0	0
0906	1	3	2	1	0	0
0907	13	30	5	7	13	5
0908	2	3	0	2	1	0
0909	2	26	3	5	14	4
0910	13	27	5	11	10	1
0911	5	9	0	4	3	2
0912	10	24	5	9	9	1
0913	8	29	9	5	15	0
0914	11	28	8	10	9	1
0915	7	20	5	5	9	1
0916	5	19	4	2	12	1

NORTH DORCHESTER

	CLIENTS	SAMPLES	LOW	MEDIUM	HIGH	V. HIGH
0917	11	34	10	13	11	0
0918	4	15	9	4	2	0
0919	4	15	1	7	7	0
0920	29	97	51	26	18	2
0921	14	31	11	7	13	0
0922	29	83	39	23	20	1
0923	4	20	18	2	0	0
0924	3	11	2	6	3	0
TOTAL	183	561	204 36.4%	154 27.4%	183 32.6%	20 3.6%

SOUTH DORCHESTER

	CLIENTS	SAMPLES	LOW	MEDIUM	HIGH	V. HIGH
1001	6	8	4	2	0	2
1002	2	9	4	4	1	0
1003	6	40	21	13	5	1
1004	20	48	16	20	12	0
1005	20	59	37	15	7	0
1006.01	3	10	0	3	7	0
1006.02	5	9	1	1	7	0
1007	3	6	3	2	1	0
1008	15	36	18	15	3	0
1009	10	27	16	2	9	0
1010.01	8	9	7	2	0	0
UNK.	7	20	7	9	4	0
1010.02	13	19	11	7	1	0
1011.01	4	7	5	2	0	0
1011.02	1	2	0	1	1	0
TOTAL	123	309	150 48.5%	98 31.7%	58 18.8%	3 1%

EAST BOSTON

	CLIENTS	SAMPLES	LOW	MEDIUM	HIGH	V. HIGH
0501	4	7	3	2	2	0
0502	4	5	1	4	0	0
0503	3	4	2	2	0	0
0504	3	5	3	1	1	0
0505	2	2	0	1	1	0
0506	2	5	0	2	1	2
0507	1	2	1	1	0	0
0508	3	8	5	2	1	0
0509	3	3	0	1	2	0
0510	5	11	4	2	5	0
0511	10	24	16	7	1	0
0512	2	19	16	2	1	0
UNK.	2	5	2	2	1	0
TOTAL	44	100	53	29	16	2
			53%	29%	16%	2%

HYDE PARK

1401.01	29	76	52	16	7	1
1401.02	12	16	15	1	0	0
1402	31	67	45	13	9	0
1403	18	31	13	15	2	1
1404	8	17	13	4	0	0
TOTAL	98	207	138	49	18	2
			66.7%	23.7%	8.7%	.9%

JAMAICA PLAIN	CLIENTS	SAMPLES	LOW	MEDIUM	HIGH	V. HIGH
1201	58	132	58	44	29	1
1202	17	48	20	17	11	0
1203	25	73	22	29	20	2
1204	65	176	59	63	52	2
1205	19	74	37	15	20	2
1206	41	125	56	40	29	0
1207	13	26	15	9	1	1
UNK.	2	8	1	3	4	0
TOTAL	240	662	268 40.5%	220 33.2%	166 25.1%	8 1.2%
NORTH END						
0301	0	0	0	0	0	0
0302	0	0	0	0	0	0
0303	3	3	2	1	0	0
0304	1	1	1	0	0	0
0305	1	1	1	0	0	0
TOTAL	5	5	4 80%	1 20%	0	0
ROSLINDALE						
1101.01	3	12	11	1	0	0
1101.02	22	56	29	17	10	0
1102	1	1	0	1	0	0
1103	13	28	16	7	4	1
1104.01	13	32	19	12	1	0
1104.02	8	23	22	1	0	0
1105.01	7	9	5	2	2	0
1105.02	7	21	2	14	2	3

ROSLINDALE	CLIENTS	SAMPLES	LOW	MEDIUM	HIGH	V. HIGH
1106.01	9	16	15	1	0	0
1106.02	14	25	15	9	1	0
TOTAL	97	223	134 60.1%	65 29.1%	20 9%	4 1.8%
ROXBURY						
0801	1	4	0	2	2	0
0802	9	41	19	14	7	1
0803	8	27	14	10	2	1
0804	3	12	9	2	1	0
0805	8	30	23	7	0	0
0806	2	6	5	0	1	0
0807	0	0	0	0	0	0
0808	1	3	3	0	0	0
0809	13	34	14	11	5	4
0810	9	22	20	1	1	0
0811	7	21	10	8	2	1
0812	7	19	4	7	8	0
0813	7	36	21	10	3	2
0814	17	83	31	23	22	7
0815	7	37	6	6	13	12
0816	3	12	10	1	1	0
0817	4	14	13	0	1	0
0818	7	27	15	8	4	0
UNK.	14	72	54	15	3	0
0819	3	7	1	3	3	0
0820	6	17	6	11	0	0
0821	2	6	6	0	0	0
TOTAL	138	530	284 53.6%	139 26.2%	79 14.9%	28 5.3%

SOUTH BOSTON	CLIENTS	SAMPLES	LOW	MEDIUM	HIGH	V. HIGH
0601	8	23	4	4	5	0
0602	4	12	5	2	5	0
0603	3	14	0	2	12	0
0604	6	16	3	8	2	3
0605	3	18	14	3	1	0
0606	0	0	0	0	0	0
0607	2	8	8	0	0	0
0608	3	7	5	0	0	2
0609	3	3	1	0	2	0
0610	3	3	1	0	1	1
0611	2	2	2	0	0	0
0612	1	1	1	0	0	0
0613	3	11	0	6	1	4
0614	2	7	5	0	1	1
UNK.	4	10	9	0	0	1
TOTAL	47	135	58 43%	35 25.9%	30 22.2%	12 8.9%
SOUTH END						
0701	12	73	49	17	7	0
0702	6	22	17	3	2	0
0703	21	50	36	8	6	0
0704	0	0	0	0	0	0
0705	12	31	8	13	10	0
0706	11	26	14	8	4	0
0707	11	42	7	25	10	0
0708	19	37	23	10	4	0
0709	7	30	19	3	8	0
0710	7	11	4	4	3	0
0711	0	0	0	0	0	0

SOUTH END	CLIENTS	SAMPLES	LOW	MEDIUM	HIGH	V. HIGH
0712	4	23	6	9	5	3
TOTAL	110	345	183	100	59	3
			53%	29%	17.1%	.9%
WEST END						
0201	13	20	18	1	1	0
0202	6	7	3	4	0	0
0203	9	20	19	1	0	0
TOTAL	28	47	40	6	1	0
			85.1%	12.8%	2.1%	
WEST ROXBURY						
UNK.	1	1	1	0	0	0
1301	17	25	19	6	0	0
1302	16	36	25	7	4	0
1303	13	22	15	3	4	0
1304.01	12	19	13	4	2	0
1304.02	11	13	12	1	0	0
TOTAL	70	116	85	21	10	0
			73.3%	18.1%	8.6%	
HARBOR ISLANDS						
1501	0	0	0	0	0	0
TOTAL, BOSTON	1521	3978	2078	1094	720	86
			(52.2%)	(27.5%)	(18.1%)	(2.2%)
TOTAL, ALL	2344	6119	2807	1761	1361	190
			(45.9%)	(28.8%)	(22.2%)	(3.1%)

TABLE 6

Emergency Lead Poisoning Areas: Average Soil Lead Content At Homes Of Lead-Poisoned Children

ELPA Number	ELPA Census Tract	PPM Lead	#Samples (N=128)
1	802/906	3420	6
2	804	674	16
3	812	2993	4
4	818	-	-
5	901	5183	3
6	901/902	458	2
7	903	672	6
8	906/913	2113	3
9	913	4322	4
10	914(e)	1445	7
11	914(w)	3563	4
12	915	1933	5
13	917/918	2262	6
14	917/920	1449	14
15	919	788	4
16	923	1848	5
17	924(sw)	642	3
18	924(n)	2645	2
19	924(se)	1392	4
20	1001	1225	2
21	1002(n)	-	-
22	1002(s)	1807	8
23	1004	1633	5
24	1005(w)	1754	5

TABLE 6 (con't)

ELPA Number	ELPA Census Tract	PPM Lead	# Samples (N=128)
25	1005(se)	14320	2
26	1011.02(n)	1651	5
27	1011.02(s)	650	1
28	1205	2332	2
Average		2006	

APPENDIX C

Estimation of Population within the Receptor Array

APPENDIX C

Estimation of Population within the Receptor Array

Procedure utilized in obtaining an estimate of total population in a 10 km radius from the proposed site of the facility (receptor array):

The region consists of Suffolk and portions of Norfolk, and Middlesex counties. U.S. Bureau of Census population data for the state of Massachusetts (1980) was used for computational purposes.

ANALYSIS

(I) Suffolk County total population data;

census code 25025, name: Suffolk, Population (1980) = 650, 142

Census tracks excluded from 10 km critical radius are 1402, 1401.02, 1304.01, 1304.02, 1301. Total populations for these tracts were 30,665 persons. Therefore, 619,477 persons reside in the target area (650, 142-30, 665).

(II) Other cities included in target area are:

Name	Population (1980 Census)
Everett	37,195
Somerville	77,372
Medford	58,076
Cambridge	95,322
Watertown	34,384
Brookline	55,062
TOTAL	**357,411**

TOTAL EXPOSED POPULATION=650,142-30,655+357,411=**976,880 persons**.

APPENDIX D

Polychlorinated Dibenzo-p-Dioxins and Dibenzofurans (PCDDs and PCDFs)

APPENDIX D

Polychlorinated Dibenzo-p-Dioxins and Dibenzofurans (PCDDs and PCDFs)

TCDD was first identified 30 years ago, when Sorge established it as the cause of industrial chloracne (cited by Ayres et al., 1985). During the 1950's, 60's, and 70's, chloracne remained the most frequent apparent consequence of human exposure to reasonably high levels of TCDD: changes in liver function, lower extremity neuropathy, and psychological abnormalities were also clinical findings associated with relatively high-level exposures to TCDD. None of the clinical literature pointed to the possibility of cancer arising from these exposures.

A. Evidence for the Carcinogenicity of PCDD/PCDF.

Laboratory Animal Bioassays.

In 1977, Van Miller et al. presented the first evidence that 2,3,7,8-TCDD could induce various tumors in Sprague-Dawley rats. This preliminary work was confirmed and extended by a more complete bioassay performed by Kociba and co-workers (1978). Although 2,3,7,8-TCDD proved to be the most potent carcinogen ever tested in rats, a clear dose-response relationship showed that it is not infinitely potent. The highest dose -- 0.1 ug TCDD/kg body weight/day -- was clearly toxic, depressing weight gains, causing changes in liver structure and function, and altering lymphoid tissues and hematologic parameters. This dose of 2,3,7,8-TCDD increased the incidence of hepatocellular carcinomas and squamous cell tumors at various sites, and oddly, decreased the incidence of tumors at five other sites, including the mammary glands and uterus. Decrements in the incidence of specific tumors meant that the total incidences of tumors -- 60, 48, and 40 tumors per 50 animals dosed with the high, medium, and low doses of 2,3,7,8-TCDD respectively -- were each actually less

than the total incidence of 138 tumors among the 85 control animals.

The intermediate dose of TCDD -- 0.01 ug/kg/day -- caused fewer and less severe toxic changes, and was tumorigenic only in that it induced benign hepatic nodules. The lowest dose -- 0.001 ug/kg/day -- resulted in no apparent toxicity or pathology. In fact, at this dose the incidence of every tumor type reported was actually lower than the spontaneous incidence observed in the controls. The data are summarized in the following two tables.

TABLE 3.3.2B

SUMMARY OF SIGNIFICANT TUMOUR INCIDENCE IN MALE SPRAGUE-DAWLEY RATS MAINTAINED ON DIETS CONTAINING 2,3,7,8-T_4CDD[a]

Time Interval of Study	Month 0-12				Month 13-24				Terminal Kill				Total			
Dose level (ug/kg/day)	Control	0.001	0.01	0.1	Control	0.001	0.01	0.1	Control	0.001	0.01	0.1	Control	0.001	0.01	0.1
Number of rats examined	5	1	0	4	65	38	46	41	15	11	4	5	85	50	50	50
Rats with Tumours/tumour-like lesions																
Tumours significantly (P<0.005) greater than controls																
Carcinoma (stratified squamous cell) hard palate or nasal turbinates	0	0	0	0	0	0	0	4	0	0	0	0	0	0	0	4[b]
tongue	0	0	0	0	0	1	1	3	0	0	0	0	0	1	1	3[b]
Adenoma of adrenal cortex	0	0	0	0	0	0	2	5	0	0	0	0	0	0	2	5[b]
Tumours significantly less than controls																
Fibroadenoma/fibroma/lipoma (subcutaneous)	1	0	0	0	8	1	4	6	1	0	1	0	10	1[c]	5	6
Acinar adenoma of pancreas	0	0	0	0	11	3	4	2	3	4	1	0	14	7	5	2[c]
Pheochromocytoma of adrenal	0	0	0	0	19	3	10	3	9	3	0	1	28	6	10	4[c]
Total tumours not significantly different from controls	2	0	0	1	56	17	23	29	28	8	2	6	86	25	25	36
Total tumours	3	0	0	1	94	25	44	52	41	15	4	7	138	40	48	60

[a] - adapted from Kociba *et al.*, 1978;
[b] - significantly greater than control ($p < 0.05$);
[c] - significantly lower than control ($p < 0.05$)

TABLE 3.3.2C

SUMMARY OF SIGNIFICANT TUMOUR INCIDENCE IN FEMALE SPRAGUE-DAWLEY RATS MAINTAINED ON DIETS CONTAINING 2,3,7,8-T_4CDD^{a}

Time Interval of Study	Month 0-12				Month 13-24				Terminal Kill				Total			
Dose level (ug/kg/day)	Control	0.001	0.01	0.1	Control	0.001	0.01	0.1	Control	0.001	0.01	0.1	Control	0.001	0.01	0.1
Number of rats examined	1	2	2	9	60	32	34	36	25	16	14	4	86	50	50	49
Rats with tumours/tumour-like lesions																
Significantly ($P < 0.05$) greater than controls																
Hyperplastic nodules -Hepatocellular	0	0	0	0	2	1	8	20	6	2	10	3	8	3	18^{b}	23^{b}
Carcinoma-Hepatacellular	0	0	0	0	0	0	0	10	1	0	1	1	1	0	1	11^{b}
Carcinoma (stratified squamous cell) of hard palate or nasal turbinates	0	0	0	0	0	0	1	4	0	0	0	0	0	0	1	4^{b}
Carcinoma (keratinizing squamous cell) of lung	0	0	0	0	0	0	0	7	0	0	0	0	0	0	0	7^{b}
Significantly less than control																
Benign tumour - uterus	0	0	0	2	16	5	7	3	12	7	4	2	28	12	11	7^{c}
Benign neoplasm - mammary gland	0	0	1	0	50	24	23	22	23	11	12	2	73	35	36	24^{c}
Carcinoma - mammary gland	0	0	0	0	5	3	1	0	6	1	5	0	11	4	6	0^{c}
Adenoma - pituitary	0	0	0	0	26	12	8	12	17	6	5	0	43	18	13	12^{c}
Total not significantly different from controls	0	0	0	0	45	12	9	31	24	12	8	0	69	24	17	31
Total tumours/tumour-like lesions	0	0	1	2	144	37	58	109	89	39	45	8	233	76	104	119

[a] - adapted from Kociba *et al.*, 1978;
[b] - significantly greater than controls ($p < 0.05$);
[c] - significantly lower than controls ($p < 0.05$)

The National Toxicology Program (1982a) tested 2,3,7,8-TCDD by gavage administration in Osborne-Mendel rats. The doses used -- 0.07 ug, 0.007 ug, and 0.0014 ug/kg/day -- were reasonably similar to those utilized by Kociba et al. (1978), and reasonably similar results were obtained. For male rats, dosed animals showed no increases in total tumors relative to controls, although the high dose and intermediate dose caused statistically significant increases in the incidence of thyroid follicular cell carcinomas or adenomas relative to vehicle (that is, sham-gavaged) controls. (But interestingly, the incidence of thyroid tumors in the untreated controls was also significantly higher than that incidence in vehicle controls) The lowest dose of TCDD resulted in no increased incidence of tumors.

For female rats, the high dose led to an increased incidence of hepatic neoplastic nodules, and to an increase in hepatic neoplastic nodules and carcinomas, when the two lesions were considered together. No significant effects were observed to result from the intermediate or low dose. The data appear in the following two tables.

TABLE 3.3.2D
SUMMARY OF SIGNIFICANT TUMOUR NUMBERS IN MALE OSBORNE-MENDEL RATS ADMINISTERED 2,3,7,8-T_4CDD BY GAVAGE[1]

	Untreated Control	Vehicle Control	Low Dose	Mid Dose	High Dose	Dose Related Response
Design Dose (ug/kg/day)	0.000	0.000	0.0014	0.007	0.07	
Number of Rats Examined	75	75	50	50	50	
Carcinoma or Adenoma-Thyroid Follicular Cell	8	1 (104)[2]	5^c (56)	8^c (99)	11^a (77)	S.D.
Fibroma - Subcutaneous	3	3 (75)	1 (107)	3 (93)	7^c (70)	S.D.
Adenoma - Adrenal-cortical cell	8	6 (87)	9 (72)	12^b (70)	9 (84)	N.S.D.
Total Primary Tumours	73	57	38	53	55	
Total Animals with Tumours	48	40	29	33	32	
Incidence of Primary Tumours	0.97	0.76	0.76	1.06	1.10	

[1] From NIH No. 82-1765 (NTP, 1982a)

[2] - time tumour first appeared (weeks)
S.D. - significant dose response.
N.S.D. - no significant dose response.
a - significantly greater than control ($p < 0.001$)
b - significantly greater than control ($p < 0.02$)
c - significantly greater than control ($p < 0.05$)

TABLE 3.3.2E

SUMMARY OF SIGNIFICANT TUMOUR NUMBERS IN FEMALES OSBORNE-MENDEL RATS ADMINISTERED WITH 2,3,7,8-T_4CDD BY GAVAGE[1]

	Untreated Control	Vehicle Control	Low Dose	Mid Dose	High Dose	Dose Related Response
Design Dose (ug/kg/day)	0.000	0.000	0.0014	0.007	0.07	
Number of Rats Examined	75	75	50	50	49	
Adenoma or Thyroid Follicular Cell	0	3 (90)	2 (105)	1 (104)	6 (95)	S.D.
Neoplastic Nodules - Liver	1	5 $(90)^2$	1 (104)	3 (106)	12^b (85)	S.D.
Neoplastic Nodules or Carcinoma - Liver	2	5 (90)	1 (104)	3 (106)	14^a (61)	S.D.
Fibrosarcoma - Subcutaneous	-	0 (-)	2 (55)	3 (58)	4^c (95)	N.S.D.
Adenoma NOS - Pituitary	0	1 (104)	5^c (76)	2 (104)	3 (99)	N.S.
Adenoma or Carcinoma Adrenal	10	11 (77)	9 (65)	5 (83)	14^c (85)	S.D.
Total Primary Tumours	76	82	62	51	90	
Total Animals with Tumours	50	54	40	36	43	
Incidence of Primary Tumours	1.01	1.09	1.24	1.02	1.84	

[1] From NIH No. 82-1765 (NTP, 1982a)

^{2} - time tumour first appeared
SD - significant dose response
N.S.D. - no significant dose response
^{a} - significantly different to control ($p < .001$)
^{b} - significantly different to control ($p < .017$)
^{c} - significantly different to control ($p < .05$)

The NTP (1982a) also bioassayed 2,3,7,8-TCDD in B6C3F1 mice. Male mice were tested at the same doses as those used for rats, while females received the relatively higher doses of 0.3 ug, 0.03 ug, and 0.006 ug/kg/day. In males, the high dose of TCDD induced a statistically significant increase in the incidence of hepatocellular adenomas or carcinomas relative to controls. No significant increases in these tumors were observed in the intermediate or low dose groups in pair-wise comparisons with the control group, although a significant dose-related "trend" did hold across all three doses.

In female mice, the high dose again caused significant increases in the incidence of combined hepatic adenomas or carcinomas. Unlike male mice, females at the high dose level also responded with increased incidences both of thyroid follicular-cell adenomas and histiocytic lymphomas. No significant increases in specific tumor types were observed in the intermediate or low-dose groups, although total tumor incidence did seem to increase across all three doses. The data are summarized in the following two tables.

TABLE 3.3.2F

SUMMARY OF NUMBER OF SIGNIFICANT TUMOURS IN MALE B6C3F1 MICE ADMINISTERED 2,3,7,8-T_4CDD BY GAVAGE[1]

	Vehicle Control	Low Dose	Mid Dose	High Dose	Dose Related Response
Design Dose (ug/kg/day)	0.000	0.0014	0.007	0.07	
Number of animals per group	75	50	50	50	
Hepatocellular Adenema or Carcinoma	15 (85)#	12 (71)	13 (79)	27* (34)	S.D.
Alveolar/Bronchiolar Adenoma or Carcinoma	10 (86)	2 (84)	4 (58)	13 (86)	S.D.
Total Primary Tumours	55	32	32	58	
Total Animals with Tumours	40	25	27	37	
INCIDENCE OF TOTAL TUMOURS	0.73	0.64	0.64	1.16	

[1] From NIH Publication No. 82-1765 (NTP, 1982a)

* Significantly different from control ($P<0.001$)

SD- Significant dose response ($P<0.004$)

# Weeks to appearance of first tumour of that type.

TABLE 3.3.2G

SUMMARY OF NUMBERS OF SIGNIFICANT TUMOURS IN FEMALE B6C3F1 MICE ADMINISTERED 2,3,7,8-T_4CDD BY GAVAGE[1]

	Vehicle Control	Low Dose	Mid Dose	High Dose	Dose Related Response
Design Dose (ug/kg/day)	0.000	0.0067	0.03	0.30	
Number of animals per group	75	50	50	50	
Hepatocellular Adenoma or Carcinoma	3 (91)#	6 (105)	6 (105)	11* (99)	S.D.
Follicular-Cell Adenoma (Thyroid)	0	3 (90)	1 (107)	5* (99)	S.D.
Histiocytic Lymphoma	18 (70)	11 (89)	13 (85)	20* 99)	S.D.
Lymphoma or Leukemia	18 (76)	12 (89)	13 (85)	20* (40)	S.D.
Subcutaneous Fibrosarcoma	1 (95)	1 (105)	1 (98)	5 (88)	S.D.
Total Primary Tumours	42	32	34	52	
Total Animals with Tumours	36	25	28	34	
INCIDENCE OF TOTAL TUMOURS	0.56	0.64	0.71	1.10	

1 - From NIH Publication No. 82-1765 (NTP, 1982a)
SD - Significant dose response ($P<0.017$)
* - Significantly different from control ($P<0.017$)
# - Weeks to appearance of first tumour of that type

Toth et al. (1979) tested 2,3,7,8-TCDD by gavage administration in Swiss/H/Riop mice. Weekly doses of 7.0, 0.7, and 0.007 ug/kg were given. In the high-dose group, significant early mortality and a 30% incidence of liver tumors were noted; in the intermediate-dose group, a 48% incidence of liver tumors occurred. (The higher tumor yield in the intermediate-dose group is presumably a function of the overall lower mortality from the other toxic effects of TCDD, as compared with the mortality observed in the high dose group. In other words, one presumes that more of the high dose animals would have developed liver tumors, had they not died prematurely due to other TCDD-related causes.)

Finally, a mixture of PCDDs have been bioassayed by the NTP (1980) in Osborne-Mendel rats and B6C3F1 mice. The mixture contained 31 percent 1,2,3,6,7,8-HexaCDD, 67 percent 1,2,3,7,8,9-HexaCDD, and 2 percent lower chlorinated DD's, including 0.04 percent tetraCDDs (unspecified isomers). Rats and male mice were given weekly doses of 5, 2.5, and 1.25 ug/kg by gavage twice per week. Doses to female mice were twice as large. High-dose female rats, and high-dose male and female mice, developed statistically significant increases in the incidences of hepatocellular adenomas, and hepatocellular adenomas or carcinomas. The PCDD mixture also induced significant liver toxicity, including degenerative changes and toxic hepatitis. The NTP reported statistically significant increases in liver tumors among intermediate- and low-dose female rats as well, although a review of the slides by three independent pathologists suggested different, lower tumor yields at these doses. The data and results of the pathologic reviews appear in the tables on the following two pages.

Table 7.1-6

Tumor Incidences in Female Osborne-Mendel Rats and Male and Female B6C3F1 Mice Given HexaCDD by Gavage for Two Years[a]

Tissue and Diagnosis	Dose Level (µg/kg-week)			
	0 (vehicle control)	1.25[b] (2.5)	2.5 (5.0)	5.0 (10)
		Female Rat		
Liver				
Neoplastic nodule or hepatocellular carcinoma	5/75[c]	10/50 p = 0.026[d]	12/50 p = 0.007	30/50 p < 0.001
		Male Mice		
Liver				
Hepatocellular adenoma	7/73	5/50	9/49	15/48 p = 0.003
Hepatocellular adenoma or carcinoma	15/73	14/50	14/49	24/48 p = 0.001
		Female Mice		
Liver				
Hepatocellular adenoma	2/73	4/48	4/47	9/47 p = 0.003
Hepatocellular adenoma or carcinoma	3/73	4/48	6/47	10/47 p = 0.004

---

a Chi-square test for trend in proportions statistically significant at α = 0.05 level for all tissues and diagnoses.

b Dose level for female rats and male mice. Number in parentheses is the dose level for female mice.

c Number of animals with tumor over number of animals examined.

d P values under treatment group incidences are from Fisher's Exact Test. Only p values less than or equal to 0.05 are reported.

Source: California Department of Health Services, 1986.

Table 7.1-7

Incidence of Liver Tumors Based on Four Separate Pathological Evaluations of Female Rats Given HexaCDD by Gavage for Two Years[a]

Pathologist and Diagnosis	Dose Level (µg/kg-week)			
	0 (control)	1.25	2.5	5.0
NTP (1980a)				
Neoplastic nodules or hepatocellular carcinoma	5/75[b]	10/50 $p = 0.026$	12/50	30/50(4)[c] $p < 0.001$
Squire (1983)				
Neoplastic nodules	1/75	4/50	7/50 $p = 0.007$	7/50 $p = 0.007$
Haberman and Schueler (Schueler 1983)				
Neoplastic nodules or hepatocellular carcinoma	---	---	---	17/50(3)[d]
Hildebrandt (1983)				
Neoplastic nodules or hepatocellular carcinoma	1/75	5/50 $p = 0.037$	7/50 $p = 0.007$	18/50(2) $p < 0.001$

---

[a] Chi-square test for trend in proportions for NTP, Squire, and Hildebrandt studies significant at $\alpha = 0.05$ level.

[b] Number of animals with tumor over number of animals examined.

[c] Number of animals diagnosed with hepatocellular carcinoma is shown in parentheses.

[d] The diagnosis for nine of the animals with neoplastic nodules is considered a matter of judgment by the pathologist.

Source: California Department of Health Services, 1986.

There have been no long-term bioassays of any of the PCDFs, so that assumptions that PCDFs are carcinogenic have yet to be tested.

In summary, then, bioassays conducted over the past decade have shown that sub-microgram per kilogram doses of 2,3,7,8-TCDD induce liver tumors in rats and mice. Other tumor types appear to be associated with relatively high-doses of TCDD, although they do not appear in both species or in both sexes. TCDD-induced tumors are accompanied by other clinical signs of TCDD induced toxicities. No-observable-effects-levels (NOELS) have been demonstrated in both rats and mice, and for all signs of chronic toxicity, including the development of tumors. The susceptibility of non-rodent species to TCDD or HexaCDD-induced tumorigenesis is not known, and PCDFs have remained untested in any species for tumorigenicity.

Epidemiology

The epidemiologic findings on TCDD and PCDDs are quite scant. Links between soft tissue sarcomas and TCDD exposures are claimed by some, disputed by others. This rare form of cancer has been diagnosed in workers involved in the production of the phenoxy herbicides 2,4-D and 2,4,5-T, but none have been directly attributed to PCDD exposure.

Since even those with occupational exposures are in fact exposed to very low levels of PCDDs, and are simultaneously exposed to complex, poorly defined mixtures of other toxicologically significant compounds, it seems unlikely that definitive epidemiologic studies can be performed. Kimbrough et al. (1984) comment:

> "Epidemiological reports have included prospective cohort studies (some with more than 3000 person-years) in which exposures have been well documented, even if not well quantified, in which no excess mortality or malignancies were observed. The few case-control reports that suggest an excess risk of cancer are compromised by their poorly documented and obviously heterogeneous exposures, uncertainty that controls were appropriately selected, and the potential for introduction of a serious error through recall bias."

Individuals living in close proximity to incinerators do not appear to suffer increases in overall or specific cancer mortality. Zack (1984) of the CDC studied cancer mortality from 1968 through 1978 for residents living within 12 census tracts that surrounded a municipal solid waste incinerator in Philadelphia. A statistically non-significant decrease of 1% in deaths from all cancers was observed relative to that expected for the general population.

Mutagenesis and Genotoxicity

2,3,7,8-TCDD has been extensively tested for mutagenicity in many *in vitro* and *in vivo* systems. In various strains of Salmonella, 2,3,7,8-TCDD has consistently yielded negative results, both with and without mammalian microsomal activation, and in strains sensitive both to base substitution mutations and to frame-shift mutations. Early works by Seiler (1973) and Hussain *et al*. (1972) in which 2,3,7,8-TCDD had tested positive in two strains of Salmonella are considered to be of questionable validity, due to high cell toxicity at concentrations of 2,3,7,8-TCDD necessary to produce positive results, and to irreproducibility in later experiments. Overall, then, 2,3,7,8-TCDD is generally accepted to be non-mutagenic in Salmonella (Commoner, 1976; McCann, 1978; Wassom *et al*., 1978; Gilbert, *et al*., 1980; Geiger and Neal, 1981; Mortelmans *et al*., 1984). The table on the following page summarizes the results of testing and the different genotypes used.

TABLE 3.3.1.1A
SALMONELLA MAMMALIAN MICROSOMAL MUTAGENICITY TEST RESULTS

	STRAIN GENOTYPE													
	G46	TA1975	TA1950	TH1530	TH1535	TA100	TA1531	TH1532	TA1537	TA1978	TA1534	TA1538	TA98	REFERENCE
Histidine mutation	G46	G46	G46	G46	G46	G46	C207	C3076	C3076	C3052	D3052	D3052	D3052	Ames et al., 1973
Lipopoly-saccharide	+	rfa	+	gal	rfa	rfa	gal	gal	rfa	rfa	+	rfa	rfa	Ibid
DNA Repair	+	+	uvrB	uvrB	uvrB	uvrB	uvrB	uvrB	uvrB	+	uvrB	uvrB	uvrB	Ibid
PKM101 (episome)	-	-	-	-	-	+	-	-	-	-	-	-	+	Ibid
							2,3,7,8-TCDD							
Spot test + S9					-			-	-			-		McCann, 1978 Wassom et al., 1978
Spot test - S9	-			-			?	+			?			Seiler, 1973
Plate + -S9	-	-	-	-		-		-	-			-	-	Gilbert et al., 1980
Plate + S9					-	-			-			-	-	Geiger + Neal 1981
Plate - S9														Ibid
Suspension assay - S9				-				T	-					Hussain et al., 1972
Fluctuation test + - S9		-	-	-	-	-		-	-	-		-	-	Gilbert et al., 1980
							O_8CDD							
Spot test - S9	-			-			-	?			?			Seiler, 1973
							Dibenzo-p-dioxin							
Plate Incorp. + - S9					-	-			-			-		Commoner, 1976

+ = positive; ? = questionable response; - = negative; blank = not tested; T = dose toxic to bacterial strain.

2,3,7,8-TCDD has also been tested for intercalating activity using calf thymus DNA _in vitro_. The results demonstrated no detectable effect on spectral or melting characteristics, suggesting that 2,3,7,8-TCDD does not intercalate (Hay, 1983).

Using _in vitro_ and host-mediated assays, Bronzetti _et al._, (1983) have reported positive results in yeast. At concentrations of 10 ug/ml, 2,3,7,8-TCDD induced a 4 fold increase in gene conversion and revertant events, in addition to moderate cell toxicity. Although 2,3,7,8-TCDD does appear to be a modest mutagen in yeast, this type of information is not typically factored into assessments of risk to human cells.

In mammalian cells, unscheduled DNA synthesis is used as an indicator of DNA damage due to exposure to genotoxic chemicals. Negative results in cultured rat hepatocyte cells (Althaus _et al._, 1982) and in cultured human cells (Loprieno _et al._, 1982) indicate that 2,3,7,8-TCDD does not induce DNA damage in mammalian cells _in vitro_.

The cell transformation ability of 2,3,7,8-TCDD has been reported by Hay (1983). In assays using baby hamster kidney fibroblasts, induction of cell transformation was demonstrated with a minimum effective dose of 25 ug/kg cell culture medium. In a more recent study using mouse embryo fibroblasts, Abernethy (1984) reported that 2,3,7,8-TCDD did not initiate cell transformation and was therefore not mutagenic in this test system.

Chemical adduction to DNA is often taken as a sign of genotoxicity. Konderosi _et al_ (1973) reported that 2,3,7,8-TCDD does not form adducts to DNA when tested _in vitro_. Poland and Glover (1979) investigated the potential of 2,3,7,8-TCDD to adduct to DNA _in vivo_. The authors reported that none of the

administered 2,3,7,8-TCDD was associated with rat liver DNA. Thus the compound does not appear to form DNA adducts.

To test the mutagenicity of chemicals on mammalian cells *in vivo*, the primary genetic endpoint observed is chromosomal damage. Although cells from a number of different species have been investigated (rat, rabbit, man, and others) findings, when positive, have been inconclusive. The table on the following page summarizes the *in vivo* genotoxic events reported to be associated with 2,3,7,8-TCDD. In its assessment of the health effects of 2,3,7,8-TCDD, the Canadian Government (Ontario Ministry of the Environment, 1985) has this to conclude about the genotoxicity of 2,3,7,8-TCDD:

> "Results of testing of 2,3,7,8-TCDD in mammalian systems have, to date, provided insufficient evidence of chromosomal damaging activity of this compound in *in vivo* systems. While a weak induction of chromosomal aberrations in rats and induction in humans have been reported in isolated studies, the predominance of evidence suggest the compound is not clastogenic."

TABLE 3.3.1.7A

IN VIVO GENOTOXIC EVENTS ASSOCIATED WITH 2,3,7,8-T_4CDD

SPECIES	ROUTE OF ADMINISTRATION	DOSE ug/kg	DOSES APPLIED (freq)	EXPER. DURATION	GENO-TOXIC EFFECT	END-POINT
1. Rat	Oral	200	1	4 weeks	+?	Multinucleated Cells
2. Rabbit	Cutaneous	?	?	?	+?	Multinucleated cells
3. Rat (male)	Gavage I.P. Gavage	10 5,10,15 20	5(daily 1 1	29 days 4 hours 29 days	- - -	chromosomal aberration
4. Rat (male) Osborne-Mendel (Female)	intubation	0.25,1,2,4 .25,5,2,4	26 bi-weekly	13 weeks	+ +	chromosomal aberrations
5. Human	?	?	?	?	-	chromatid and chromosomal aberration
6. Human 4 adults 13 children	?	?	?	?	-	chromosomal aberrations
7. Human	?	?	acute chronic	?	-	chromosomal aberrations
8. Human 8 adults	? ?	? ?	acute chronic	3 years	-(yr1) +(yr3) +(yr1)	decrease in D-group chromosome satellite
9. Human 15 adults	?	?	?	6-16 mos.	-	chromosomal aberration S.C.E.

In summary, 2,3,7,8-TCDD is not mutagenic in microbial mutagenicity tests. It does not intercalate, adduct or otherwise damage DNA, nor does it appear to be clastogenic or induce chromosomal aberrations in animals or humans. 2,3,7,8-TCDD is an unconfirmed mutagen and possesses cell transformation ability in only one *in vitro* mammalian system. It is generally concluded that 2,3,7,8-TCDD is not a classical mutagen (Ontario Ministry of the Environment, 1985), and it seems clear that the carcinogenicity of TCDD is not mediated *via* mutagenicity.

B. Acute Toxicity and Scaling Factors

2,3,7,8-TCDD is the most well characterized PCDD, but it represents a very small portion of the total PCDD content of incinerator emissions. The Hart report (1984), for example, estimates that 2,3,7,8-TCDD makes up less than 0.73% of the total PCDDs, and less than 0.076% of the combined total PCDDs and PCDFs predicted in the subject facility emissions.

This means that estimates of carcinogenic risk due to complex mixtures of PCDDs and PCDFs in emissions are difficult to calculate, since there is simply a lack of data on the carcinogenicity of the other congeners and isomers. This lack of data has forced risk assessors to look to measurements of acute toxicities, which are somewhat more readily available, and to information on biochemical mechanisms of action, in order to judge how carcinogenic an isomer *might* be, relative to the carcinogenicity of 2,3,7,8-TCDD.

One of the most striking characteristics of the acute toxicity of 2,3,7,8-TCDD is its interspecies variability. A more than 5000-fold range in sensitivity separates the most sensitive (guinea pig) from the least sensitive (hamster) species. The table following summarizes acute mammalian toxicity.

In addition to the wide variability in lethal dose, there is also a wide variation in the histopathology of the toxic response among different mammals (Poland and Knutson, 1982). Chloracne, for example, is a clinical marker for 2,3,7,8-TCDD exposure in humans, but a similar condition is observed in only one test mammal, the monkey. With respect to liver lesions, rabbits and rats appear to be more sensitive than monkeys, and possibly humans. A summary of the interspecies differences in physiological response to TCDD exposure is found on the second table following.

TABLE 3.2.2A
ACUTE MAMMALIAN TOXICITY OF 2,3,7,8-T_4CDD

Species, Strain	Sex	Route of Exposure	Single Dose LD_{50} (ug/kg body wt.)	References
Guinea pig, Hartley	M	oral	0.6	Schwetz *et al.*, 1973
	F	oral	2.1	McConnell *et al.*, 1978a
Rat, Sherman	M	oral	22	Schwetz *et al.*, 1973
	M	i.p.*	60	
	F	oral	45	
	F	i.p.	25	
Rat, Sprague Dawley	M	oral	60	Beatty *et al.*, 1978
Weanling	M	oral	60	
	F	oral	25	
Rat, Charles River	Mixed	oral	100	
Rat, Porton	F	oral	90	
Monkey, Rhesus	F	oral	ca.70	McConnell *et al.*, 1978b
Rabbit	Mixed	oral	115	Schwetz *et al.*, 1973
		dermal	272	
		i.p.	252	
Mouse, C57B1/Sch	M	oral	114	Schwetz *et al.*, 1973
Mouse, C57B1/6fh	M	oral	284	McConnell *et al.*, 1978a
Mouse, C57BL/6J		i.p.	132	Olson *et al.*, 1983
Mouse, DBA/2J		i.p.	620	
Mouse, B6D2F/J		i.p.	300	
Dog, Beagle	Mixed	oral	ca.200-300	Schwetz *et al.*, 1973
Hamster, Golden (Syrian)	Mixed	oral	1157	Olson *et al.*, 1980b
	Mixed	i.p.	3000	Henck *et al.*, 1981
	M	oral	5051	Gupta *et al.*, 1973

* i.p. - interperitoneal

TABLE 3.2.2B

ACUTE TOXICITY AND HISTOPATHOLOGY OF 2,3,7,8-T_4CDD OR RELATED CHLORINATED AROMATIC HYDROCARBONS: SPECIES DIFFERENCES

SPECIES	ACUTE TOXICITY LD_{50} ug/kg-bw	CHRONIC TOXICITY ug/kg-day	WASTING SYNDROME	HYPERPLASIA AND/OR METAPLASIA							HYPOPLASIA, ATROPHY OR NECROSIS			OTHER	
				(1)	(2)	(3)	(4)	(5)	(6)	(7)	(A)	(B)	(C)	(A1)	(A2)
Monkey	70		x	++	+	++	++		++		+	+	+	+	+
Guinea Pig	0.6- 2.1		x	0		++					+	+	+	0	0
Cow			x	+		++	+			+	+	+	+	0	0
Rat	22- 190	.01-0.1	x	0		0		++	0		+		+	++	0
Mouse	114- 300	0.26-0.4	x	0		0	++		0		+	+	+	+	+
Rabbit	115- 272		x			++			+					++	
Chicken	25- 50		x								+	+	+	+	++
Hamster	1157-5051		x	0	++						+			+	+
Dog	200- 300		x												

x demonstrated effect   0 no effect   +,++ demonstrated effect ranked in severity   Blank - no data

(1) Gastric mucosa
(2) Intestinal mucosa
(3) Urinary tract
(4) Bile duct and/or Gall Bladder
(5) Lung (focal alveolar)
(6) Skin (chloracne)
(7) Skin (X-disease)

(A) Thymus
(B) Bone Marrow
(C) Testicle

(A1) Liver Lesions
(A2) Oedema

Amalgam from Table 3.2.2A and Poland and Knutson, 1982

Different species develop very different symptoms upon exposure to TCDD. This an especially difficult and confounding factor in the process of attempting to extrapolate dose/response relationships in test animals to humans. While exposure to lethal levels of TCDD results in a characteristic "wasting syndrome", no single symptom, indeed, no single chemical lesion or target tissue has been implicated as the exact cause of death.

The other PCDD congeners and isomers are all less toxic than 2,3,7,8-TCDD. Congeners and isomers that carry chlorine substitution in at least three of the four lateral ring positions -- positions 2,3,7,and 8 -- appear to be maximally toxic, although even among this group of closely related PCDD isomers, toxic potencies vary by from 100 to 10,000 fold. The other, less toxic PCDD isomers congeners are some 15,000 to 150,000 less potent than 2,3,7,8-TCDD. Octa-CDD, for example, has a reported LD50 of greater than 1 gram per kg in mice and rats (Schwetz *et al.*, 1973). In general, the negligibly toxic congeners, namely, the mono-, di-, tri, and octa-CDDs, are not considered in the assessment of carcinogenic risk of incinerator emissions. Kociba and Schwetz (1982) have reviewed the acute toxicities of the various PCDD congeners in the guinea pig and the mouse. The following table summarizes their findings.

TABLE 3.6.2A

ACUTE TOXICITIES AND AHH INDUCTION EFFICIENCIES OF PCDD

		ACUTE TOXICITY		AHH INDUCTION			
				in vivo			in vitro
		Guinea Pig LD_{50} ug/kg	Mouse LD_{50} ug/kg	Chicken Embryo ED_{50} ug/egg	C57BL/6 Mouse ED_{50} ug/kg	DBA/2B Mouse ED_{50} ug/kg	Rat Hepatoma EC_{50} ug/L
DD			>50,000	-			-
M_1CDD				-			-
D_2CDD	1,3			-			-
	1,6			-			-
	2,3			-			-
	2,7		>2,000,000	-			-
	2,8	>300,000	847,000,000	6.3			-
T_3CDD	1,2,4			1.4			
	2,3,7	29,444	>3,000	1.1			330
T_4CDD	1,2,3,4						-
	1,2,3,8						201
	1,3,6,8						-
	1,3,7,8			1.3			29
	2,3,7,8	2	283.7	0.007	0.27	3.9	0.12
	1,3,7,9						
P_5CDD	1,2,3,7,8	3.1	337.5				2.0
	1,2,4,7,8	1,125	>5,000	0.012			45,000
H_6CDD	1,2,3,4,7,8	72.5	825	0.16			3.0
	1,2,3,6,7,8	70-100	1,250	0.55			13
	1,2,3,6,7,9						32,000
	1,2,3,7,8,9	60-100	>1,440	0.01			18
	1,2,4,6,7,9			-			-
H_7CDD	1,2,3,4,6,7,8	>600		1.61			59
	1,2,3,4,6,7,9						1,600
O_8CDD	1,2,3,4,5,6,7,8		>4,000,000				9,200

Adapted from Kociba and Schwetz, 1982a.

- no induction at dose tested.

The PCDFs have yet to be as thoroughly evaluated as the PCDDs for toxic potential. Acute toxicity of only 3 PCDFs have been tested in guinea pigs, mice, and rhesus monkeys (Moore _et al_., 1979). The major symptoms of acute toxicity parallel those of the PCDDs. A summary of the reported findings appears below.

PCDF	ANIMAL SPECIES	LD50
2,3,7,8-TCDF	guinea pig	7 ug/kg
	monkey	300 - 100 ug/kg
	rat	>1000 ug/kg
	mouse	>6000 ug/kg
2,3,4,7,8-PCDF	guinea pig	<10 ug/kg
2,3,4,6,7,8-HCDF	guinea pig	120 ug/kg

(Abstracted from Ontario Ministry of the Environment, 1985.)

In addition to acute toxicity, some biochemical endpoints have been used to estimate the relative potencies of PCDDs/PCDFs. The ability of TCDD (as well as the other PCDDs) to induce the expression of oxidative enzyme systems -- primarily aryl hydrocarbon hydroxylase (AHH) -- has been well characterized. This enzyme system oxidizes many xenobiotic compounds, including chemical carcinogens. While AHH acts to convert PCDDs into _less_ toxic metabolites, it acts upon many other chemical carcinogens to activate them into _more_ toxic and carcinogenic forms. The isomers which are most potent inducers of AHH activity have chlorines at all four of the lateral ring positions (2,3,7,8), and induction potency has been shown to correlate closely with _acute_ toxicity _in vivo_ (Poland and Knutson, 1982; Poland _et al_., 1979; Bradlaw and Casterline, 1979). It is generally accepted that PCDD (or PCDF) binds to a protein receptor in the cytosol as a prerequisite to induction of AHH activity.

A parallel situation exists with the PCDFs: the compounds which are the most acutely toxic are also the most potent inducers of AHH. The following table outlines the hepatic AHH induction efficiencies of the various PCDF isomers, while enzyme induction for the PCDDs appears in the table on page 72.

TABLE 3.6.6A

HEPATIC ARYL HYDROCARBON HYDROXYLASE INDUCTION EFFICIENCY BY PCDF ISOMERS

		in vivo Level of AHH activity relative to control				*in vitro* Calculated EC_{50}	
		rat liver		mouse liver		rat hepatoma cells	
ANALOGUE	ISOMER	a)	b)	c)	d)	e) (ug/plate)	f) (ug/flask)
DF						-(8.4)	
M_1CDF	4						2035
D_2CDF	2,6					-(11.9)	14620
	2,8	-(10,000)					9360
	2,3						519
T_3CDF	1,3,6						687
	2,4,6					126.5(P)	
	1,3,8						5267
	2,3,4						41
	2,4,8		-(5)				
	2,6,7						760
	2,3,8*					0.8	676
T_4CDF	1,4,6,8					-(16.1)	
	1,2,4,8						3672
	1,3,6,7	-(5,000)	-(5)				
	1,3,6,8	-(5,000)					
	2,3,4,6						404
	2,4,6,8					-(16.1)	
	1,2,7,8*	5(1,000)					
	2,3,4,8*						1217
	2,3,6,7*	5(5,000)	-(5)	0.8(30)	1.3(30)		
	2,3,6,8		1.3(5)			20.8(P)	316
	2,3,7,8*	11(5,000)	4(5)**	1.0(30)	13.6(30)**	0.004	1.2
P_5CDF	1,2,4,6,7						111
	1,2,4,6,8	-(5,000)					
	1,2,4,7,9						12.8
	1,2,3,4,8*						71
	1,2,3,6,7*		-(5)	0.8(30)	1.0(30)		
	1,2,4,7,8		1.4(5)			1.5	36
	1,2,6,7,8*	6(1,000)	-(5)				
	1,3,4,7,8					0.09	
	2,3,4,6,7*	12(1,000)	-(5)	0.8(30)	2.7(30)		
	1,2,3,7,8*	7(10,000)	-(5)	1.1(30)	6.9(30)**		0.9
	2,3,4,7,8*	9(1,000)	2.0(5)**	0.9(30)	7.6(30)**		0.09
H_6CDF	1,2,3,4,6,7*	6(1,000)	-(5)	0.9(30)	1.2(30)		
	1,2,4,6,7,8						15.9
	1,2,3,4,7,8*	8(10,000)	1.3(5)	0.9(30)	1.6(30)		0.13
	1,2,3,6,7,8*		-(5)				0.55
	2,3,4,6,7,8*						0.26
H_7CDF	1,2,3,4,6, 8,9					1.4	
T_4CDD	2,3,7,8*		7.0(5)	10.9(30)	21.0(30)	0.0005	0.002

a) Yoshihara *et al.*, 1981 (Wistar Rat - male - liver)
b) Nagayama *et al.*, 1983a (Wistar Rat - male - liver)
c) Nagayama *et al.*, 1983b (DDD Mouse - liver)
d) Nagayama *et al.*, 1983b (AKR Mouse - liver)
e) Bradlaw and Casterline, 1979 (Rat Hepatoma Cell Culture)
f) Bandiera *et al.*, 1984 (Rat Hepatoma Cell Culture)
- indicates no induction at dose tested, (ug/kg body weight or ug/container)
* chlorine substitution in 3 lateral positions (2, 3, 7 or 8) and one vicinal hydrogen or chlorine substitution in all 4 lateral positions
** indicates significant induction ($p < 0.01$)
(P) indicates projected EC_{50}

In lieu of adequate toxicity and carcinogenicity data, then, it has become common practice of risk assessors to use the degree to which liver enzymes are induced in test animals as a benchmark of potential carcinogenicity for the PCDDs and PCDFs. Although this may be an appropriate starting point for ranking such potentials, several caveats must be addressed.

First, AHH induction and receptor affinities are quite similar across various species, while the LD50 values vary widely across these species. The binding affinity and concentrations of the cytosolic receptor in the guinea pig, mouse, rat, and hamster, for example, are very similar despite the greater than 5,000 fold difference in the TCDD LD50 between guinea pig and hamster (Poland and Knutson, 1982). This should preclude the use of enzyme induction efficiencies as absolute values for toxic potency (Poland and Knutson, 1982).

Second, not every compound which induces AHH activity is a carcinogen; many known non-carcinogens are AHH inducers. It is also the case that many carcinogens do not induce AHH activity. It must be acknowledged that this is not a well understood mechanism, and there are some very important limitations in equating the ability to induce AHH activity with carcinogenic potential.

In attempting to assess the net carcinogenic potential of a mixture of PCDDs and PCDFs, several criteria and assumptions are used to develop scaling factors for each of the compounds. Approaches which cope with the attendant uncertainties are generally divided into 3 areas, based on:

1. assumptions of enzyme inducing abilities relative to the most potent inducer, 2,3,7,8-TCDD;

2. assumptions regarding the structure-activity relationships (which are that toxicity is directly related to chlorination at least three of the 2,3,7,and 8 lateral ring positions, and other congeners are ignored); and
3. actual data from biological monitoring, comparing activity of the mixture to 2,3,7,8-TCDD.

Obviously experimental data should be preferred over theoretical exercises. The following table outlines the different approaches to dealing with complex mixtures of PCDDs and PCDFs.

TABLE 3.6.7A
2,3,7,8-T$_4$CDD TOXIC EQUIVALENTS

E.G. NO.	SAMPLE	ANALYTICAL METHOD QUANTITATED	ASSUMPTIONS OR BIOANALYTICAL METHOD USED	X-FOLD TOXICITY OF THE MIXTURE ABOVE THE MEASURED VALUE FOR 2,3,7,8-T$_4$CDD	REFERENCE
1	Particulate emission from Resource Recovery Facility	Total T$_4$CDD's and 2,3,7,8-T$_4$CDD as 7% of total T$_4$CDDs	Assumed all T$_4$CDD's as toxic as 2,3,7,8-T$_4$CDD.	14X	(Redford, 1981)
2	Flue gas emission from municipal incinerator	2,3,7,8-T$_4$CDD & Isomer Groups T$_4$CDD T$_4$CDF P$_5$CDD P$_5$CDF H$_6$CDD H$_6$CDF H$_7$CDD H$_7$CDF O$_8$CDD O$_8$CDF	ASSUMED ENZYME INDUCTION ABILITY Assumed relative toxicity based on following microsomal enzyme induction ability: 2,3,7,8-T$_4$CDD 1.0 Other T$_4$CDD 0.01 T$_4$CDF 0.1 P$_5$CDD 0.1 P$_5$CDF 0.1 H$_6$CDD 0.1 H$_6$CDF 0.1 H$_7$CDD 0.01 H$_7$CDF 0.01 O$_8$CDD - O$_8$CDF - Assume entire isomer group has the same induction ability as the most potent inducer in that group (Conservative). Used above factors as weighting factors to convert analytically measured isomer group masses to 2,3,7,8-T$_4$CDD equivalents.	60X	(Swiss Federal Office for Environmental Protection, 1982)
3	Fly ash extract	2,3,7,8-T$_4$CDD	RECEPTOR BIOASSAY METHOD Measured dose-dependant ability of extract to displace radiolabelled 2,3,7,8-T$_4$CDD from the cytosol receptor protein; EC$_{50}$ values i.e., the amount of unlabelled ligand to displace one-half of the [3H]-T$_4$CDD are used for comparing mixtures to 2,3,7,8-T$_4$CDD.	45X	(Hutzinger, et al., 1981)

TABLE 3.6.7A (Cont'd)

<table>
<tr><th>E.G. NO.</th><th>SAMPLE</th><th>ANALYTICAL METHOD QUANTITATED</th><th>ASSUMPTIONS OR BIOANALYTICAL METHOD USED</th><th>X-FOLD TOXICITY OF THE MIXTURE ABOVE THE MEASURED VALUE FOR 2,3,7,8-T$_4$CDD</th><th>REFERENCE</th></tr>
<tr><td>4</td><td>Fly ash extract</td><td>2,3,7,8-T$_4$CDD</td><td>ENZYME INDUCTION METHOD
Measured dose-dependent ability of extract to induce the aryl hydrocarbon hydroxylase (AHH) enzyme system. EC$_{50}$ values (i.e. the amount of half-maximal induction) are measured by two techniques (i.e. AHH & EROD) and are used for comparing mixtures to 2,3,7,8-T$_4$CDD.</td><td>8X (AHH)
26X (EROD)</td><td>(Sawyer, et al., 1983)</td></tr>
<tr><td>5</td><td>Flue gas emission from municipal reactor</td><td>2,3,7,8-T$_4$CDD & Total Homologues
T$_4$CDD T$_4$CDF
P$_5$CDD P$_5$CDF
H$_6$CDD H$_6$CDF
H$_7$CDD H$_7$CDF</td><td>ASSUMPTION OF POLANDS'S HYPOTHESIS re Toxicity
Assume toxicity of PCDD's and PCDF's related to substitution in three of four of the 2,3,7 and 8 positions.
These congeners were considered to be of concern; the rest were ignored:
i.e.
<table>
<tr><th></th><th>Total # of isomers</th><th># of Toxic isomers</th></tr>
<tr><td>T$_4$CDD</td><td>22</td><td>5 (incl. 2,3,7,8-T$_4$CDD)</td></tr>
<tr><td>P$_5$CDD</td><td>14</td><td>7</td></tr>
<tr><td>H$_6$CDD</td><td>10</td><td>7</td></tr>
<tr><td>H$_7$CDD</td><td>2</td><td>1</td></tr>
<tr><td>T$_4$CDF</td><td>38</td><td>8</td></tr>
<tr><td>P$_5$CDF</td><td>28</td><td>14</td></tr>
<tr><td>H$_6$CDF</td><td>16</td><td>12</td></tr>
<tr><td>H$_7$CDF</td><td>4</td><td>2</td></tr>
</table>
Further assume:
- 2,3,7,8-T$_4$CDD = 3.3% of total T$_4$CDD.
- All "Toxic" compounds have a toxicity of 10% compared to 2,3,7,8-T$_4$CDD.
- Isomers are equally distributed in each isomer group.
Calculation:
$$\left[\begin{array}{l}\text{2,3,7,8-T}_4\text{CDD toxic}\\ \text{equivalent of an}\\ \text{isomer group}\end{array}\right] = \frac{\left[\begin{array}{l}\text{Analytically}\\ \text{measured mass of}\\ \text{isomer group}\end{array}\right]}{\left[\text{\# isomers in group}\right]} \times \left[\begin{array}{l}\text{\# of "toxic"}\\ \text{isomers}\end{array}\right] \times 0.1$$</td><td>80X</td><td>(Hutzinger, et al., 1983)
(Grant, 1977)</td></tr>
</table>

The following table outlines the scaling factors and basis for derivation used by 10 other authors and/or agencies. Some are based on rather "arbitrary" criteria. But it is clear that those which look at the largest number of factors in their consideration of relative potencies are the most desirable, e.g., the methods of the FDA and Ontario. As an example of the depth to which this type of analysis can go, the following seven page insert is a compilation of the assumptions made by the Canadian Government in developing their scaling or "prorating" factors for the PCDD and PCDF isomer groups. The Canadian document (Ontario Ministry of the Environment, 1986) states that "development of the relative toxicity factors of the various PCDD and PCDF isomers compared to 2,3,7,8-T4CDD is based on a review of current research data."

Some Approaches to Estimating Relative Toxicities of PCDDs and PCDFs

Basics/Compound	Hart 1984	SWISS	Grant Olie	New York State	Ontario	FDA	CA	EPA 1982	EPA Current Recommended
(Basis)	Enzyme Toxicity	Enzyme	Toxic Equiv. factor	LD50	various effects	various effects			Carcin.
Mono tru di	0	0	0	0	0	0	0	0	0
tri	0.01	0	0	0	1	0	0	0	0
2378-TCDD	1	1	1	1	1	1	1	1	1
other TCDDs	0.01	0.01	0	0	0.01	0	0	1	0.01
2378-PeCDDs	0.1	0.1	0.1	1	1	0	1	0	0.2
other PeCDDs	0.1	0.1	0	0	0.01	0	0	0	0.002
2378-HxCDDs	0.1	0.1	0.1	0.03	1	0.02	1	0	0.04
other HxCDDs	0.1	0.1	0	0	0.01	0.02	0	0	0.0004
2378-HpCDDs	0.01	0.01	0.1	0	1	0.005	1	0	0.001
other HpCDDs	0.01	0.01	0	0	0.01	0.005	0	0	0.00001
OCDD	0	0	0	0	0	0.00001	1	0	0
2378-TCDFs	0.1	0.1	0.1	0.33	0.02	0	1	0	0.1
other TCDFs	0.1	0.1	0	0	0.0002	0	0	0	0.001
2378-PeCDFs	0.1	0.1	0.1	0.33	0.02	0	1	0	0.1
other PeCDFs	0.1	0.1	0	0	0.0002	0	0	0	0.001
2378-HxCDFs	0.1	0.1	0.1	0.01	0.02	0	1	0	0.01
other HxCDFs	0.1	0.1	0	0	0.0002	0	0	0	0.0001
2378-HpCDFs	0.01	0.1	0.1	0	0.02	0	1	0	0.0001
other HpCDFs	0.01	0	0	0		0	0	0	0.00001
OCDF	0	0	0	0	0	0	0	0	0

Source: Environmental Protection Agency

Assumptions which lead to the different proposed toxicity prorating factors for each PCDD or PCDF isomer group are as follows:

Dibenzo-p-dioxin (DD) and Dibenzofuran (DF)

- parent molecules
- no LD_{50} data
- inactive in *in vitro* AHH induction assay
- no carcinogenic response in lifetime studies of rats and mice fed 10,000 ppm DD in their diet
- not considered toxic.

M_1CDDs

- 2 isomers
- no LD_{50} data
- no *in vitro* AHH induction data
- no fetal anomalies in litters of Wistar rats fed 2,000 ug 2-M_1CDD/kg/day during gestation days 6 to 15. Comparable NOEL for 2,3,7,8-T_4CDD is 0.03 ug/kg/ day suggesting that M_1CDDs are 67,000-fold less teratogenic than 2,3,7,8-T_4CDD.
- toxicity assumed less than or comparable to O_8CDD (see below).

D_2CDDs

- 10 isomers .
- LD_{50} (guinea pig) for 2,8-D_2CDD is 150,000-fold less toxic than 2,3,7,8-T_4CDD
- 2,8-D_2CDD not active in *in vitro* AHH induction test (25,400-fold less active than 2,3,7,8-T_4CDD)
- slight myocardial oedema in fetuses of Wistar rats fed 500 ug 2,7-D_2CDD/kg/day on gestation

days 6 to 15. Comparable LEL for 2,3,7,8-T_4CDD in rats is 0.125 ug/kg/day suggesting that 2,7-D_2CDD is 4,000-fold less teratogenic than 2,3,7,8-T_4CDD.

- suggestive carcinogenic effect in male B6C3F1 mice fed 10,000 ppm 2,7-D_2CDD in diet (estimated at 1,000 ug 2,7-D_2CDD/kg/day). Comparable LEL for 2,3,7,8-$_4$CDD in male B6C3F1 mice is 0.07 ug/kg/day suggesting that 2,7-D_2CDD is about 14,000-fold less carcinogenic than 2,3,7,8-T_4CDD.
- assume D_2CDD isomers have a toxicity of 0.1% (i.e. 0.001) compared with 2,3,7,8-T_4CDD.

T_3CDDs

- 14 isomers, 2 potentially toxic
- LD_{50} 2,3,7-T_3CDD is greater than 11-fold (mouse) to 15,000-fold (guinea pig) less toxic than 2,3,7,8-T_4CDD
- *in vitro* AHH induction activity (2,3,7-T_3CDD) is 157-fold to 2,750-fold less active than 2,3,7,8-T_4CDD
- no teratology or carcinogenicity data
- assume T_3CDD isomers have a toxicity of 1.0% (i.e. 0.01) compared with 2,3,7,8-T_4CDD

T_4CDDs

- 22 isomers, 5 (including 2,3,7,8-T_4CDD) potentially toxic
- toxicity of 2,3,7,8-T_4CDD well established
- other T_4CDD isomers treated separately from 2,3,7,8-T_4CDD
- no LD_{50} data for other T_4CDD isomers
- *in vitro* AHH induction, most active isomer (1,3,7,8-T_4CDD) is 185-fold to 240-fold less active than 2,3,7,8-T_4CDD

- no fetal or postnatal effects in progeny of rats treated with 800 ug 1,2,3,4-T_4CDD/kg/day during gestation day 6 to 15. Comparable NOEL for 2,3,7,8-T_4CDD is 0.03 ug/kg/ day suggesting 1,2,3,4-T_4CDD is 27,000-fold less toxic than 2,3,7,8-T_4CDD.
- no carcinogenicity data for other T_4CDD isomers
- treat 2,3,7,8-T_4CDD separately (100% toxicity)
- assume other T_4CDD isomers have a toxicity of 1% (i.e. 0.01) compared with 2,3,7,8-T_4CDD

P_5CDDs

- 14 isomers, 7 potentially toxic
- LD_{50} (1,2,3,7,8-P_5CDD) 1.2-fold (mouse) to 1.5-fold (guinea pig) less toxic than 2,3,7,8-T_4CDD
- *in vitro* AHH induction (1,2,3,7,8-P_5CDD) is 17-fold less active than 2,3,7,8-T_4CDD
- no teratology or carcinogenicity data
- assume P_5CDD isomers have a toxicity of 10% (i.e. 0.1) compared with 2,3,7,8-T_4CDD

H_6CDDs

- 10 isomers, 7 potentially toxic
- LD_{50} (1,2,3,4,7,8-H_6CDD) 3-fold (mouse) to 36-fold (guinea pig) less toxic than 2,3,7,8-T_4CDD
- *in vitro* AHH induction (1,2,3,4,7,8-H_6CDD) 25-fold to 80-fold less active than 2,3,7,8-T_4CDD
- the NOEL for embryotoxicity and teratogenicity for mixtures of H_6CDDs in rats is 0.1 ug/kg/day during fetal development, the comparable NOEL for 2,3,7,8-T_4CDD is 0.03 ug/kg/day suggesting that mixtures of HCDDs are 30-fold less teratogenic than 2,3,7,8-T_4CDD
- the NOEL for carcinogenicity for a mixture of 1,2,3,6,7,8-H_6CDD and 1,2,3,7,8,9-H_6CDD in rats is 1.25 ug/kg/day, the comparable NOEL for

2,3,7,8-T_4CDD is 0.001 ug/kg/day suggesting that mixtures of H_6CDDs are 1,250-fold less carcinogenic than 2,3,7,8-T_4CDD

- assume H_6CDD isomers have a toxicity 10% (i.e. 0.1) compared with 2,3,7,8-T_4CDD

$\underline{H_7\text{CDDs}}$

- 2 isomers, both potentially toxic
- LD_{50} (1,2,3,4,6,7,8-H_7CDD) guinea pig > 300-fold less toxic than 2,3,7,8-TCDD
- _in vitro_ AHH induction (1,2,3,4,6,7,8-H_6CDD) 500-fold less active than 2,3,7,8-T_4CDD
- no teratology or carcinogenicity data
- assume H_7CDD isomers have a toxicity of 1.0% (i.e.) 0.01 compared with 2,3,7,8-T_4CDD

$\underline{O_8\text{CDD}}$

- 1 isomer
- LD_{50} (rat and mouse) >500,000-fold less toxic than 2,3,7,8-T_4CDD
- _in vitro_ AHH induction >46,000-fold less active than 2,3,7,8-T_4CDD
- no fetal anomalies in rats fed 100,000 ug O_8CDD/kg/day during fetal development. Comparable NOEL for 2,3,7,8-T_4CDD is 0.03 ug/kg/day suggesting that O_8CDD is at least 3,000,000-fold less teratogenic than 2,3,7,8-T_4CDD.
- no carcinogenicity data
- assume O_8CDD has a toxicity of 0.01% (i.e. 0.0001) compared with 2,3,7,8-T_4CDD

$\underline{M_1\text{CDFs}}$

- 4 isomers
- no LD_{50} data
- _in vitro_ AHH induction (4-M_1CDF) about 1,000,000-fold less active than 2,3,7,8-T_4CDD
- no teratology or carcinogenicity data

- assume M_1CDF isomers have a toxicity less than or equivalent to O_8CDD (see above)

D_2CDFs

- 16 isomers
- no LD_{50} data
- *in vitro* AHH induction (2,3-D_2CDF) 260,000-fold less active than 2,3,7,8-T_4CDD
- no teratology or carcinogenicity data
- assume D_2CDF isomers have a toxicity less than or equal to O_8CDD (see above)

T_3CDFs

- 28 isomers, 4 potentially toxic
- no LD_{50} data
- *in vitro* AHH induction (2,3,8-T_3CDF) 1,600-fold less active than 2,3,7,8-T_4CDD
- no teratology or carcinogenicity data
- assume by analogy with T_3CDDs that T_3CDFs have a toxicity 1% (i.e. 0.01) compared with 2,3,7,8-T_4CDD

T_4CDFs

- 38 isomers, 8 potentially toxic
- LD_{50} data (2,3,7,8-T_4CDF) one third as toxic as 2,3,7,8-T_4CDD
- *in vitro* AHH Induction (2,3,7,8-T_4CDF) 8-fold less active as 2,3,7,8-T_4CDD
- *in vivo* AHH induction (2,3,7,8-T_4CDF) about 60% as active as 2,3,7,8-T_4CDD
- NOEL for teratogenicity in C57BL/6 mice is 10 ug/kg/day during fetal development, comparable NOEL for 2,3,7,8-T_4CDD is 0.1 ug/kg/day suggesting that 2,3,7,8-T_4CDF is 100-fold less teratogenic than 2,3,7,8-T_4CDD
- no carcinogenicity data

- assume T$_4$CDF isomers have a toxicity of 50% (i.e. 0.5) compared with 2,3,7,8-T$_4$CDD

P$_5$CDFs

- 28 isomers, 14 potentially toxic
- LD$_{50}$ (2,3,4,7,8-P$_5$CDF) one third as toxic as 2,3,7,8-T$_4$CDD
- *in vitro* AHH induction (2,3,4,7,8-P$_5$CDF) 45-fold less toxic than 2,3,7,8-T$_4$CDD
- *in vivo* AHH induction (2,3,4,7,8-P$_5$CDF) about 30% as active as 2,3,7,8-T$_4$CDD
- no teratology or carcinogenicity data
- assume that P$_5$CDF isomers have a toxicity of 50% (i.e. 0.5) compared with 2,3,7,8-T$_4$CDD

H$_6$CDFs

- 16 isomers, 12 potentially toxic
- LD$_{50}$ (2,3,4,6,7,8-H$_6$CDF) 60-fold less toxic than 2,3,7,8-T$_4$CDD
- *in vitro* AHH Induction (1,2,3,4,7,8-H$_6$CDF) 65-fold lass active than 2,3,7,8-T$_4$CDD
- no teratology or carcinogenicity data
- assume that H$_6$CDF isomers have a toxicity of 10% (i.e. 0.1) compared with 2,3,7,8-T$_4$CDD

H$_7$CDFs

- 4 isomers, 2 potentially toxic
- no LD$_{50}$ data
- *in vitro* AHH induction (1,2,3,4,6,8,9-H$_7$CDF) 120,000-fold less active than 2,3,7,8-T$_4$CDD
- no teratology or carcinogenicity data
- assume by analogy with H$_7$CDD isomers that H$_7$CDF isomers have a toxicity of 1% (i.e. 0.01) compared with 2,3,7,8-T$_4$CDD

O_8CDF

- one isomer
- no toxicity or enzyme induction data available
- assume by analogy with O_8CDD that O_8CDF has a toxicity of 0.01% (i.e. 0.0001) compared with 2,3,7,8-T_4CDD

In summary, the following prorating factors relative to 2,3,7,8-T_4CDD are proposed:

TABLE 3.6.7C

ESTIMATED RELATIVE TOXICITY OF PCDD AND PCDF ISOMERS TO 2,3,7,8-T_4CDD

Isomer Groups	Toxicity factor relative to 2,3,7,8-T_4CDD
DD	non toxic
M_1CDD	0.0001
D_2CDD	0.001
T_3CDD	0.01
T_4CDD*	0.01
P_5CDD	0.1
H_6CDD	0.1
H_7CDD	0.01
O_8CDD	0.0001
DF	non toxic
M_1CDF	0.0001
D_2CDF	0.0001
T_3CDF	0.01
T_4CDF	0.5
P_5CDF	0.5
H_6CDF	0.1
H_7CDF	0.01
O_8CDF	0.0001

* excluding 2,3,7,8-T_4CDD

Other studies have estimated TCDD equivalency using extracts of fly-ash in bioassay systems. These assays do not use actual flue gas emissions -- so the PCDDs and PCDFs present in the extracts may not be representative of actual emissions -- but these assays are likely to generate more accurate results than estimates which are more theoretically derived. Two direct measures of enzyme induction have been made using fly ash extracts: the experimental results of the two studies suggest much lower estimates of 2,3,7,8-TCDD equivalents for fly ash than those calculated by Hart.

C. Ingestion of Contaminated Soil

Kimbrough (published in Kimbrough et al., 1984) states that the age-weighted average -- which is dominated by the 10 grams of soil per day assumed to be ingested for 1-1/2 to 3-1/2 year olds -- for a 70 year lifetime is calculated to be 0.41 grams of soil per day. See the following table for CDC soil ingestion assumptions. Upon what did Kimbrough base her assumptions? Kimbrough et al. (1984, p. 73) state only that the age-specific estimates were "based on work done studying lead uptake from contaminated soils," but no citations are given to that work.

In fact, all of the work on soil ingestion of which we are aware suggests much lower amounts of soil ingestion, both by children and by adults. For example, Barltrop (1973) and Lepow et al. (1974, 1975) estimate that children between the ages of 2 years and 6 years typically ingest 100 mg of soil per day, and that persons younger and older than this ingest no significant quantities of soil. Duggan and Williams (1977) agree that only those aged 2 years through 6 years ingest significant quantities of soil, and they posit the daily amount at 50 mg of soil. The National Research Council, in a study on environmental lead, suggests a soil ingestion figure of 40 mg per day. Bellinger et

al. (1986) estimate soil ingestion at 20 mg per day. Finally, Hawley (1985) calculates that adults could consume 57 mg of soil daily, those aged 6 through 18 years could consume 21 mg, those aged 2 through 6 years could ingest 90 mg daily, and those younger than 2 years would consume negligible quantities of soil.

Table 2.

CDC Assumption: Ingestion of Dirt*

Age Group		Soil Ingested	% Lifetime Uptake
0 - 9	Mo	0 g/Day	0%
9 - 18	Mo	1 g/Day	2.6%
1.5 - 3.5	Yr	10 g/Day	70.0%
3.5 - 5	Yr	1 g/Day	5.2%
5 - 70	Yr	100 mg/Day	22.6%

*Adjusted for seasonal variations

Source: Paustenbach *et al.*, 1986.

There is, then, no published study that supports the 10 gm per day estimate for soil ingestion by toddlers. The highest number that has been suggested by anyone other than Kimbrough is 100 mg per day for toddlers, with considerably lower estimates for soil ingestion by other age groups. At a recent symposium (Workshop on Assessing Health Risks from Chemical Contaminants in Soil, May 8, 1986, Andover, MA) Paustenbach suggested that an ingestion estimate of 100 mg soil per day for toddlers was both supported by experimental data and still appropriately "conservative". Since neither Kimbrough nor any of the other speakers or attendees of the conference objected, this appears to represent the new consensus. Calabrese and co-workers at the University of Massachusetts are currently conducting another study designed to quantify soil ingestion by children. Results are anticipated within one year (Calabrese, personal communication, July 3, 1986).

Umbreit et al (1986) report that the bioavailability of TCDD ingested from contaminated soil is less than 0.5%, as confirmed by tissue analysis. A review of experimental parameters of this and other studies shows that a number of physical, chemical and biological variables can reduce the bioavailability of TCDD (Paustenbach et al., 1986). For soil that children might actually ingest, gastrointestinal absorption and bioavailability of 10% is appropriately conservative (Paustenbach et al., 1986).

D. Inhalation

In estimating the risk due to inhaled TCDD adsorbed to particulates, Hart assumed that an average of 15 m^3 of air is exchanged per day, and 75% of inhaled particles are retained in the lungs, with a 100% bioavailability. Hart pointed out that this is probably a conservative estimate. The estimate of 15 m^3 of air is one typically used in risk assessment. In a review of

the literature, however, Paustenbach et al. (1986) find that it is much more accurate to assume that, of the total respired particles, 50% are retained in the lungs and 50% are deposited in the upper airways and are swallowed. From the preceding discussion on bioavailability across the gastrointestinal tract, it follows that only 10% of the TCDD adsorbed to the swallowed particulates are likely to be bioavailable. If PCDDs and PCDFs are present on fly ash that is deposited deep inside the lung, it may be reasonable to assume that 100% of these compounds will be bioavailable. But it is important to note that none of the laboratory animal bioassays have tested PCDD/PCDF via inhalation, so that there is absolutely no data on its availability and toxicity across the lung.

Thus, if one (conservatively) assumes 100% bioavailability of TCDD on particulates retained in the lungs and 10% availability of swallowed TCDD, then total bioavailability of inhaled TCDD is closer to 55%.

E. Dermal Contact and Absorption

The amount of TCDD absorbed from dermal contact depends on the amount of contaminated material adhering to skin, the frequency and duration of exposure, and the bioavailability across the skin. Hart assumed direct contact of 0.55 grams of soil per day over a 70 year lifetime with an absorption rate of up to 10%. This appears to be an excessively conservative estimate. A study by Roels et al. (1980), for example, shows that about 0.5 mg/cm^2 of material adheres to a child's hands after playing in and around the home. A far more reasonable estimate of lifetime exposure would be obtained by multiplying this figure by the surface area. This alternative dermal exposure assumption is outlined in the following table from Paustenbach et al. (1986).

Table 5.

Exposure Assumptions Used in Case Calculations

Route	CDC	Alternate
Oral	• 0.21 g/day ingested • 30% absorption	• 0.0028 g/day ingested • 10% absorption
Dermal	• 0.21 g/day of soil on the skin • Dose is weighted by age over lifetime • 1% absorption	• 0.5 mg soil/cm² skin • 1400 cm² exposed surface • 8 hr/day, 90 days/yr • 1% absorption
Inhalation	• 0.14 mg/m³ • 15 m³/day • 100% in lungs	• Total suspended particulates is 0.075 mg/m³ • 15 m³/day • Of particles inhaled, assume 50% ingested and 50% in lungs

Source: Paustenbach, et al., 1986.

The bioavailability rate of 10% assumed by Hart is based on a study by Poiger et al. (1980) in which dermal application of TCDD dissolved in polyethylene glycol resulted in 9.3% absorption. Since TCDD is bound extremely tightly to fly ash and variably but generally strongly bound to soil (Paustenbach et al., 1986; Umbreit et al., 1986), and since it becomes less bioavailable with time and weathering, a figure of 1% would represent a far more reasonable estimate of bioavailability (Paustenbach et al., 1986).

F. Mechanisms and Biological Implications

What, then, do biological considerations of PCDDs suggest for quantitative risk assessment? The most obvious possibility is that very low levels of PCDDs may not be carcinogenic at all--neither to rats nor to humans. Typically, chemicals are tested (by the National Toxicology Program, for example) at only two positive levels. If there are carcinogenic responses at the high dose (relative to those observed in control animals), there are generally also elevated incidences in the low dose group. In general, then, one cannot demonstrate a NOAEL for chemical carcinogens.

But 2,3,7,8-TCDD, and the tested mixtures of PCDDs, appear to be in a different category. The most relied upon rat bioassay --Kociba et al., 1978 -- used three dose levels, and found no carcinogenic effects at the low and intermediate doses. Very similar results were obtained in the NTP bioassays of TCDD. One cannot be certain, of course, that these very low doses are truly noncarcinogenic, since an effect that is not apparent in groups of 50 rats might well have made itself apparent upon testing in 500 rats. On the other hand, TCDD appears to be a tumor promoter, not an initiator. Since the "one-hit" model of carcinogenesis does not appear to apply to promoters, it is

reasonably likely that there actually _is_ a threshold dose for TCDD, below which no excess risk of tumorigenesis exists. Importantly, this is in contradistinction to the case for strong, "classical," initiating carcinogens -- such as dimethylnitrosamine -- which are demonstrably carcinogenic at all tested doses and in all tested species.

The point, then, is that while 2,3,7,8-TCDD is indeed the most potent animal carcinogen known -- inducing tumors at doses of 0.1 ug/kg/day -- it is not infinitely potent. Noncarcinogenic doses do seem to exist, and might be predicted to exist on mechanistic grounds. If this is true for rodents, it is probably also true for humans. This is because at least one type of rodent tumor response -- the hepatocellular carcinoma -- appears increasingly to be an irrelevant endpoint for the purpose of human carcinogenic risk assessment (International Expert Advisory Committee to the Nutrition Foundation, 1983). Some strains of mice, in particular, display an exceptionally high _spontaneous_ incidence of hepatomas, apparently as the result of activated cellular oncogenies in liver DNA (Fox and Watanabe, 1985). Humans, in contrast, develop liver tumors only very rarely, and seem to do so primarily in the presence of a hepatitis virus.

It seems that some experts now regard TCDD as a threshold-displaying promoter. This consensus includes the Canadian government, many in the Western European scientific community, and the US Food and Drug Administration (Paustenbach _et al_., 1986). The following, for example, is the Ontario Ministry of the Environment's (1985) overall view on TCDD:

> "Review of extensive toxicological data indicates that 2,3,7,8-T_4CDD is not a classical mutagen and appears to cause tumours in rodents by an indirect mechanism. The staff committee and the Ontario Scientific Advisory Committee concluded that a threshold exists for tumour incidence and consequently 2,3,7,8-T_4CDD will not cause cancer in humans at levels below the threshold found in animal studies.

> Based on reliable chronic animal studies and extensive but inconclusive human epidemiological data, it is recommended that a threshold-safety factor approach be used to develop a maximum allowable daily intake.
>
> The proposed standard recommends an umbrella maximum allowable daily intake of 2,3,7,8-T_4CDD or its toxic equivalent from all exposure pathways based on no observable effect levels from rodent cancer bioassays and an explicit safety factor of 100.
>
> The recommended maximum allowable daily intake for 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-T_4CDD) or its toxic equivalent of PCDDs and PCDFs is 10 picograms/kilogram of body weight/day for humans."

The assumptions used in the Hart analysis, on the other hand, are based on those of the CDC and EPA; to wit, that TCDD has a linear dose response curve at low doses, despite an abundance of evidence to the contrary. Thus, it is highly likely that the estimated risks are much overstated, as has been acknowledged by the CDC in Kimbrough et al. (1984) and in recent presentations by Kimbrough.

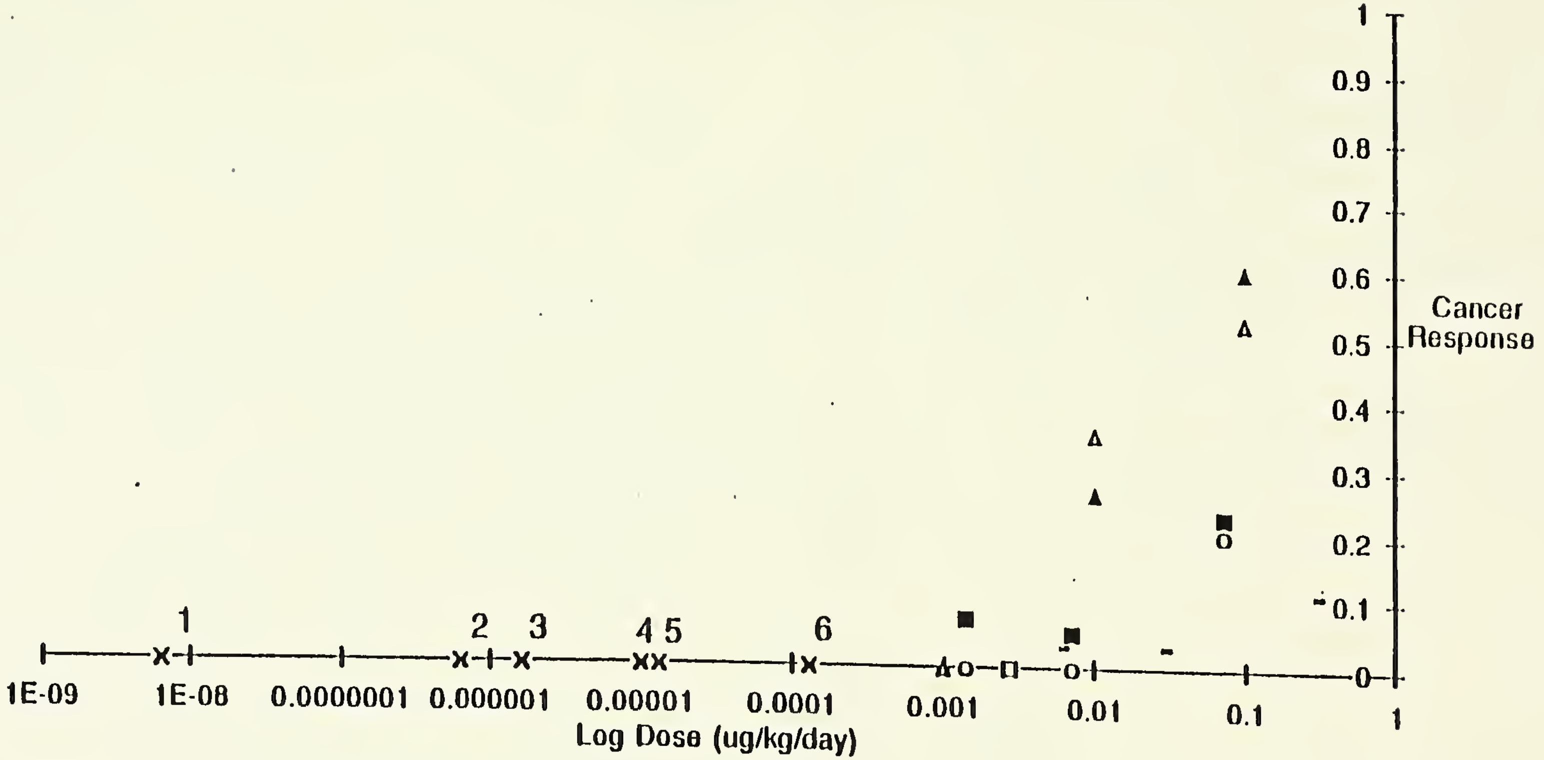
EXPERIMENTAL DATA

Reference	Endpoint/Animal Studied	Dose (ug/kg/day)	Response
NTP, 1982	Liver ca., male mice	Control	0.11
		1.430e-03	0.18
		7.143e-03	0.16
		7.143e-02	0.34
NTP, 1982	Liver ca., female mice	Control	0.01
		5.714e-03	0.04
		2.857e-02	0.04
		2.857e-01	0.13
NTP, 1982	Liver ca., female rats	Control	0.07
		1.430e-03	0.02
		7.143e-03	0.06
		7.143e-02	0.29
Kociba *et al.*, 1978	Total tumors, rats	Control	0.10
		1.000e-03	0.06
		1.000e-02	0.36
		1.000e-01	0.71
Squire's reading of Kociba *et al.*, 1978		Control	0.19
		1.000e-03	0.16
		1.000e-02	0.54
		1.000e-01	0.70
Innes *et al.*, 1969	Tumors, mice	2.700e-03	0.00

PREDICTED DOSE RESPONSE

Reference	Dose (ug/kg/day)	Response
USEPA, 1984	6.400e-09	1.0e-06
CDC, 1982	6.326e-07	1.0e-06
Hart, 1984	1.600e-06	2.5e-06
Ontario Ministry of the Environment, 1985	1.000e-05	1.0e-06
FDA, 1981	1.300e-05	1.0e-06
Paustenbach, 1986	1.300e-04	1.0e-06

Log Dose vs. Cancer Response In Population



Predicted Dose-Response

x^1 USEPA, 1984
x^2 CDC, 1982
x^3 Hart, 1984
x^4 Ontario Ministry of the Environment, 1985
x^5 FDA, 1981
x^6 Paustenbach, 1986

Experimental Data

■ Liver ca., male mice, NTP, 1982
▲ Total tumors, rats, Kociba et al., 1978
△ Total tumors, rats, Squire's reading of Kociba et al., 1978
o Liver ca., female rats, NTP, 1982
- Liver ca., female mice, NTP, 1982
□ Tumors, mice, Innes et al., 1969

APPENDIX E

Summary of Ambient Trace Elements Concentration (ug/m^3) Measured at Southampton Street, Boston

TABLE 1

SUMMARY OF AMBIENT
TRACE ELEMENTS CONCENTRATIONS (ug/m^3)
MEASURED AT SOUTHAMPTON ST., BOSTON

		Concentrations (ug/m^3)	
Element	Year	24-Hour	Annual
Arsenic (As)	1981	0.0028	*
	1982	-	-
	1983	0.0056	0.0035
	1984	0.0068	0.0029
	1985	0.0067	0.0033
Beryllium (Be)	1981	0.00131	*
	1982	0.00041	0.00023
	1983	0.00003	*
	1984	0.00003	0.00003
	1985	0.00003	0.00003
Barium (Ba)	1981	0.49	*
	1982	0.77	0.09
	1983	0.11	*
	1984	0.03	0.02
	1985	0.11	0.09
Cadmium (Cd)	1981	0.005	*
	1982	0.003	0.001
	1983	0.002	*
	1984	0.002	0.001
	1985	0.002	0.001

TABLE 1 (Continued)

		Concentrations (ug/m^3)	
Element	Year	24-Hour	Annual
Chromium (Cr)	1981	0.012	*
	1982	—	—
	1983	0.0052	*
	1984	0.0052	0.0052
	1985	0.011	0.0054
Cobalt (Co)	1981	0.0033	8
	1982	0.0025	0.0010
	1983	0.0018	0.0009
	1984	0.0021	0.0009
	1985		
Copper (Cu)	1981	0.101	*
	1982	0.107	0.063
	1983	0.181	*
	1984	0.370	0.121
	1985	0.088	0.060
Iron (Fe)	1981	1.54	*
	1982	2.35	0.99
	1983	1.71	*
	1984	2.78	1.03
	1985	2.13	1.08
Lead (Pb)	1981	0.65	*
	1982	0.94	0.44
	1983	0.48	*
	1984	0.77	0.26
	1985	0.46	0.18

TABLE 1 (Continued)

Element	Year	Concentrations (ug/m^3) 24-Hour	Annual
Manganese (Mn)	1981	0.020	*
	1982	0.042	0.021
	1983	0.020	*
	1984	0.054	0.022
	1985	0.020	0.020
Nickel (Ni)	1981	0.046	*
	1982	0.026	0.018
	1983	0.038	*
	1984	0.048	0.016
	1985	0.049	0.013
Vanadium (V)	1981	0.16	*
	1982	0.09	0.036
	1983	0.11	*
	1984	0.14	0.039
	1985	0.12	0.028
Zinc (Zn)	1981	0.40	*
	1982	0.65	0.109
	1983	0.15	0.086
	1984	0.17	0.084
	1985	0.15	

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* Insufficient data to determine annual average.